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VOL. LXVI MAY, 1956 No. 5

A STUDY OF THE ROLE OF CERTAIN FACTORS IN THE DEVELOPMENT OF SPEECH AFTER LARYNGECTOMY: 1. TYPE OF OPERATION; 2. SITE OF PSEUDOGLOTTIS; 3. COORDINATION OF SPEECH WITH RESPIRATION.†

Part 3: Coordination of Speech With Respiration.†

EVELYN Y. ROBE, Ph.D.,*
PAUL MOORE, Ph.D.,**
ALBERT H. ANDREWS, JR., M.S., M.D.,***
and
PAUL H. HOLINGER, M.S., M.D.,****
Chicago, Ill.

Loss of the vocal function as a result of laryngectomy involves not only removal of the sound producing mechanism but also exclusion of the continued use of the trachea and lungs to supply air for audible speech. Except in the case of the laryngectomized speaker who employs the reed type of

† This paper, presented in three parts, is based on the research associated with "A Study of the Role of Three Factors in the Development of Speech after Laryngectomy: Type of Operation, Site of Pseudoglottis, and Coordination of Speech with Respiration," an unpublished Ph.D. dissertation (Northwestern University, 1954) by Evelyn Y. Robe. Parts 1 and 2 appeared in the March and April, 1956, issues of The Laryngoscope.

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* Research Fellow in the William and Harriet Gould Foundation and Lecturer in Otolaryngology, Northwestern University Medical School.

** Director of Voice Research Laboratory, Northwestern University, Evanston, Ill.

*** Attending Broncho-Esophagologist and Director, Respiration Laboratory, St. Luke's Hospital, Chicago, Ill.

**** Attending Broncho-Esophagologist, St. Luke's Hospital, Chicago, Ill.

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artificial larynx, the process of pulmonary respiration is separate from that of phonic respiration.

This changed relationship between breathing and speaking frequently presents discouraging difficulties to the individual who is learning to use a substitute voice. It also provides what is perhaps the greatest point of controversy with regard to theory and methods of teaching post-laryngectomy speech: *i.e.*, whether air is inspired through the tracheostomy to the lungs at the same time that air is drawn orally into the vicarious chamber, and whether air is expired from the lungs as the oral air is used in speaking.

The literature reveals a sustained interest in investigation of the problem and nearly every article which discusses teaching methods includes strong recommendations for synchrony or asynchrony of the two processes, as the case may be.

The results of a study by Stern in 1920 of the pulmonary and phonic respiration of a large number of laryngectomized subjects, "reinforced by a great number of cases examined by Schilling", are reported by Kallen as follows:

> *1.* Types of phonic respiration in laryngectomized persons can definitely be classified.
>
> *2.* Two main types of phonic respiration seem to predominate: In one, the process of breathing is normal; in the other, renewed respiration occurs with each word, often with each syllable. In this type, the interference with the new speech mechanism by ordinary vital pulmonary respiration seems to persist.
>
> *3.* A number of forms, both mixed and transitional, fall between the two main types.
>
> *4.* In a great number of cases, the pneumograph provides a clue to the location of the vicarious air chamber and the pseudoglottis. The lower these two structures lie, the more the thoracic element is likely to play a part in the new way of speaking. This follows if one or the other component of respiration serves as an aid in "ex-

pression" of air necessary to activate the pseudoglottis.

5. The type of phonic respiration in the laryngectomized person may change with time and come to a certain stability only after many years. The change may be connected with the migration of the air chamber and with such factors as concern facilitation of neuronic pattern, etc.[5]

On the basis of his observation, Stern recommended avoidance of synchrony between pulmonary respiration and the learned process of air intake and use for speech. He believed that when the two occur together, not only is the vocal sound likely to be obscured by friction sounds at the tracheostomy as the air passes through, but coughing may also result. He urged special attention to this factor during the early stages of vocal therapy.[5]

A case of synchrony between respiration and speech in a laryngectomized person was described by Burger and Kaiser in 1925, and reported in Kallen's article. Their conclusion was based upon analysis of pneumograph recordings of respiratory movements at the nasal entrance and in the upper thoracic, gastric and abdominal regions.[5]

Kallen also referred to Froeschels, who described a patient in 1928 whose respiration and speech processes were synchronous and who was greatly aided in his efforts to obtain air for speech by the normal respiratory movement of the thorax and diaphragm. Graphic recording of the movements showed that the inspiratory and expiratory phases of speech corresponded with the movements of normal respiration during speech. When the patient was examined Roentgenologically, two phases of the speech act were observed. During the first, the diaphragm descended and at the same time the esophagus was filled with air. The second phase consisted of the rising of the diaphragm and expiration, while the esophageal walls contracted, expelling the air in speech.

Other observations regarding the action of the diaphragm during production of substitute voice were summarized by Kallen as follows:

> The general view is that the diaphragm plays a much greater part in the new speech mechanism than is supposed. In a certain sense the two reservoirs for air interfere functionally. Fluoroscopy shows peculiar jerklike movements of the diaphragm, which in selected cases obviously serve to compress the distal end of the esophagus or the gastric air bubble proper, thus aiding in the expulsion of air. That these movements have no connection with vital respiratory function is proved by the fact that they are seen to occur also when the tracheal cannula is shut off. It would follow that the diaphragm functions as an auxiliary in the new speech process, but otherwise than in normal speech.[8]

The literature reveals that the period from 1930-50 was marked by great interest in the development of programs for teaching post-laryngectomy speech. Nearly all such programs included recommendations regarding breathing and speech coordinations, although in most cases, no data were offered to support the belief that one method was superior to another.

Kallen, in 1934, recommended the reconditioning of phonic respiration as the patient's first task, even though it meant dissociating "the behavior patterns of a lifetime". The dissociation was necessary, he thought, because the old respiration habits would hinder production of the new voice and cause fatigue. Alternative methods to achieve the reconditioning were suggested: either the patient could be taught to hold the thorax in the position which follows inspiration and, while holding the position, to take in air for speech; or, the patient could take in air for speech after the lungs had been emptied of breath. He added that the latter method might be easier, but in either case it was necessary to continue to practice until conscious control of the process had been achieved.[8]

In 1936, Morrison and Fineman recommended that after the process of substitute voice had been explained, the next step was "to have the patient begin to practice the swallowing of air, always between breaths". He was to be instructed to

swallow as much air as possible, without moving the chest wall. As an aid to the process, he might place a finger tip over the tracheal tube "to remind himself that he must not breathe in or out" while swallowing air.[14] This method of dissociation evidently corresponds to the second method advocated by Kallen as given above.

Levin, in 1940, reported the following data on the "characteristics of esophageal speech, studied and compared with those of normal speech in a phonetics laboratory by spirometry and other physiologic procedures:

> *1.* Thoracic action for each syllable and breathing movements for phrasing are the same as those used in normal speech.
>
> *2.* The normal person draws air into the esophagus with the mouth open; the laryngectomized person learns to close the mouth and use the compressive movements of swallowing to force the air into the esophagus quickly.
>
> *3.* Three or four syllables can be said with one intake of esophageal air, or roughly, about 1 cc. of air for each syllable is required.
>
> *4.* The pseudoglottis must open frequently and easily for intake of air and must contract when syllables are being formed.
>
> *5.* The movements of the sternum and epigastrium are more vigorous than in the normal person; therefore, in the beginning, students of esophageal speech tire easily, and become dizzy and have other transient symptoms.[10]

As an aid in overcoming difficulty in dissociating "reflex respiratory movements from the newer mechanism", Gatewood in 1944, advocated that patients be instructed to close the tracheal opening with a finger and then "attempt to insufflate air into the throat during the act of chest expansion". Because chest expansion has always been associated with inspiration, however, it would appear that Gatewood is really

recommending association of the two processes rather than dissociation.[4]

In a discussion of a post-laryngectomy rehabilitation program in 1947, Howie also recommended "dissociation of respiration from aspiration of air into the gullet." Otherwise, the noise of air entering and leaving the trachea would mask the person's speech. Howie added that a patient using this type of speech can produce three or four syllables on one intake of air and probably uses 1 cc. of air for each syllable.[6]

Jackson and Jackson, in their program for "teaching a patient to develop articulate speech without a larynx," advocated the following instruction:

> The patient should be taught to inhale air into his esophagus by the expansion of his thorax in the ordinary breathing way, but at the moment of inspiration, the cannula must be obstructed with the finger. The air not being permitted to enter through the trachea will enter through the mouth or nose and be drawn into the esophagus.[7]

The method is based on the theory that when air is first inspirated, then swallowed, inspiration and deglutition are dissynchronized through being made to occur consecutively. One of the method's chief advantages is that "it forestalls the acquisition of the undesirable habit of air intake between short words or between syllables of longer words."[7]

Koepp-Baker advises the speech clinician to make a direct attack upon "the tendency to breathe through the tracheotomy tube during the esophageal voice production. This results in air turbulency at the orifice of the tube and obscures much of the speech." The physiological basis for such advice is as follows:

> In normal speech controlled expiration is an unconscious process. The cerebral cortex integrates the total synergy of respiration, valvulation at the larynx and the chewing, sucking movements in the mouth for normal speech production. When the total speech pattern is modified by loss of the larynx, and the esophagus and

> pharynx are used for phonation this totality must be fractionated and respiration must be inhibited."

The French phonologist, Tarneaud, stresses the importance of impressing upon the laryngectomized patient at the beginning of the treatment that the customary lung breathing must not be resumed for producing the new voice. He recommends teaching the patient to take a breath, swallow, and then to breathe out quickly. Immediately after swallowing, the sound is to be produced.[11]

Holding the breath while belching air is described by Gardner as one of the most important techniques for laryngectomized speakers to learn. The reason for its importance, he says, is that it permits the speaker "to use the abdominal wall and chest muscles to exert pressure with the greatest efficiency. If air is breathed out while trying to belch, the decreasing volume of the chest cavity tends to lessen the tension and pressure for belching."[3]

Mason, in 1950, expressed the opinion that because "the walls of the esophagus move directly as a result of movements of the thoracic cage," air can be "inspired" into the esophagus for esophageal voice by inflating the chest as in ordinary inspiration. The air is returned by raising the intrathoracic pressure and simultaneously saying syllables and words. Deliberate variations in pressure divide the syllables from each other and are independent from respiration. Mason explains that this is to avoid the noise of air blowing through the tracheal opening. She also describes associated abdominal movements which may occur at the same time, and which were formerly thought to be responsible for the separation of syllables and words. Research by Marland in 1949, however, showed that they were merely accessory since, "during a sustained conversation such movements, as detected by a hand placed on the abdomen, are found to fade away, although the voice continues unaltered in power."[12]

The literature reveals that those investigators who advocate synchrony between pulmonary and phonic respiration are decidedly in the minority. To the list of its proponents, the names

of Burger, Kaiser and Froeschels having been mentioned earlier; only Stetson and Moolenaar-Bijl are to be added.

Stetson employed apparatus specifically designed to make an objective and qualitative analysis of post-laryngectomy speech. The three forms chosen for study were: the buccal whisper, voice produced with an artificial larynx, and esophageal voice. His findings in regard to the last include the following:

> *1.* The chest action for each syllable and the breathing movements for phrasing are those of normal speech.
>
> *2.* Only a little air is taken into the upper part of the esophagus; from 2 to 5 cc.
>
> *3.* This intake is made frequently and rapidly with the mouth and velum closed; it often fuses with the movement of the consonant. Intake of air every few syllables is the one thing contrary to ordinary speech which the patient has to learn.[15]

As a result of his observations, Stetson offered an explanation as to why ordinary expiratory movements for normal speech are unsuited to esophageal speech:

> Intelligible speech cannot be made with an air supply exerting continuous pressure on the normal larynx or a substitute larynx, although that is a common assumption. The air pressure must fall to zero between the small groups of syllables, and often between syllables, and a separate pressure pulse must be made for each syllable. The one available mechanism for syllable production is the normal respiratory mechanism. Records of excellent esophageal speech show that the subject breathes just as in normal speech.[15]

Stetson concluded that a small supply of air frequently renewed could be controlled more efficiently by the original speech movements of the chest and that phrasing and accentua-

tion would sound more natural as a result. His visual records showed that "the syllable grouping, phrasing, and accentuation are produced by just such expiratory movements as in normal speech; the only difference lies in the fact that the trachea is open and the movements are somewhat more vigorous to compensate."[16]

The practice of gulping a large amount of air for substitute voice was strongly opposed by Stetson. He believed that such an intake results in continuous pressure in the esophagus contrary to the situation in normal speech and makes the utterance "hasty, forced, belching in character."[16]

Moolenaar-Bijl, in 1951, confirms Stetson's findings. As the basis for her recommendation that the natural coordination of phonation and respiration should be maintained, she expressed the opinion that physiologically and psychologically it seems the most economical way.[15]

In the course of her investigations, Moolenaar-Bijl found that phonation always appeared to be coupled with expiration. Two types could be distinguished, according to whether the swallowing or sucking technique was used. When spirometrical curves were made of the speech of the patient who used the swallowing method, it was found that the mouth of the esophagus closed after phonation, which coincided with expiration, and remained closed until the next swallow, which occurred synchronously with inspiration. Observation of the esophageal curve when the patient was not speaking revealed absence of movement. This was confirmed by X-rays which showed no air in the esophagus while the patient was silent.

Analysis of spirometrical curves of the speech of a patient who used the sucking technique led Moolenaar-Bijl to conclude that a parallelism exists between the tracheal and esophageal air movements. In speech as well as in quiet respiration, the curves of the trachea and the esophagus travelled up and down together. When X-ray photographs were made of this patient, the esophagus was observed to be filled with air before and during speech. In the process of speaking, air was pushed out of the esophagus with pulsatile movements in synchrony with syllables. Air was replenished between two respirations with little sucking movements of the tongue or lips, which were

sometimes identical with the articulation of initial plosives.[12]

The results of an experimental study by Anderson, in 1950, of some of the elements of esophageal speech proved to be inconclusive for either synchrony or asynchrony of the speech and respiration processes. A tape recorder and pneumopolygraphs were used to make simultaneous recordings at the mouth, thorax and abdomen. Analysis of the recordings revealed the following characteristics:

> Although great variation was noted from speaker to speaker, three relationships are apparent with reference to intake of air into the mouth and movements of the abdomen and thorax: *1.* Fixation of the respiratory movements of the abdomen and thorax takes place during the intake of air into the esophagus. *2.* Intake of air into the mouth is neither significantly synchronous nor asynchronous with inspiratory movements of the abdomen and thorax. *3.* Generally speaking, the abdomen and thorax exhibit synchrony of gross patterns if the record is inspected as a whole, but this synchrony does not exist if the tracings are compared for small detail.[1]

The foregoing review has shown that the majority of investigators are of the opinion that dissynchronization of breathing and speech in the laryngectomized speaker is desirable or necessary, while a minority believe that synchronization is preferable. Examples of both undoubtedly exist, and it is highly probable that some speakers shift from one to the other. It is also possible that some of those who recommend that the laryngectomized speaker learn to dissociate the two processes do not believe that the dissociation should be continued after the new speech is well-established.

The question of whether synchrony or asynchrony between breathing and phonation should be taught in post laryngectomy speech therapy is of fundamental importance to all those attempting to teach or to learn esophageal speech.

To study this relationship between phonic and pulmonary

respiration, two complementary procedures were employed. *One used a graphic recording process; the other, motion picture photography.* Twenty-three laryngectomized persons who were classified as fair, good, or superior speakers were selected

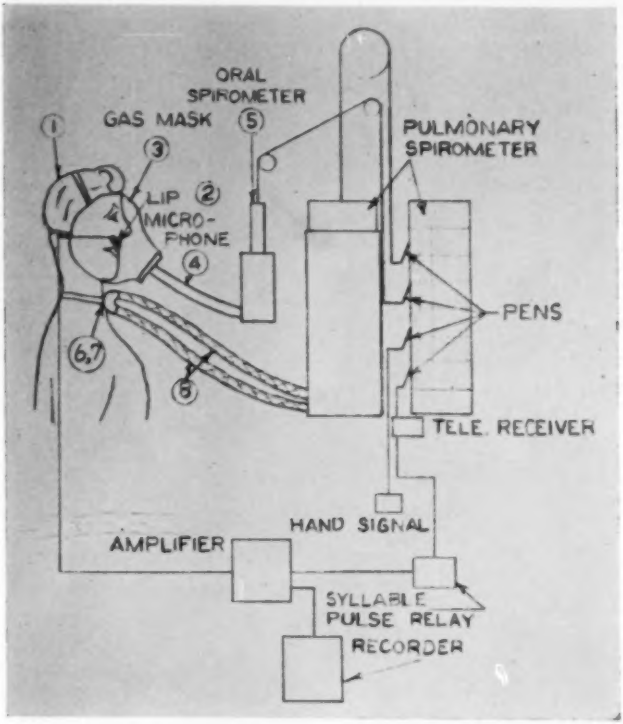

Fig. 1. Diagram of apparatus used to record pulmonary and phonic respiration.

as subjects for both parts of the procedure. The techniques in each part are reported separately and in enough detail to enable others to duplicate or extend the work if desired.

Part I: The first procedure involved the modification of equipment ordinarily used for studying the normal respira-

tory function. A description of the equipment follows, accompanied by a diagram (see Fig. 1), and photograph (see Fig. 2).

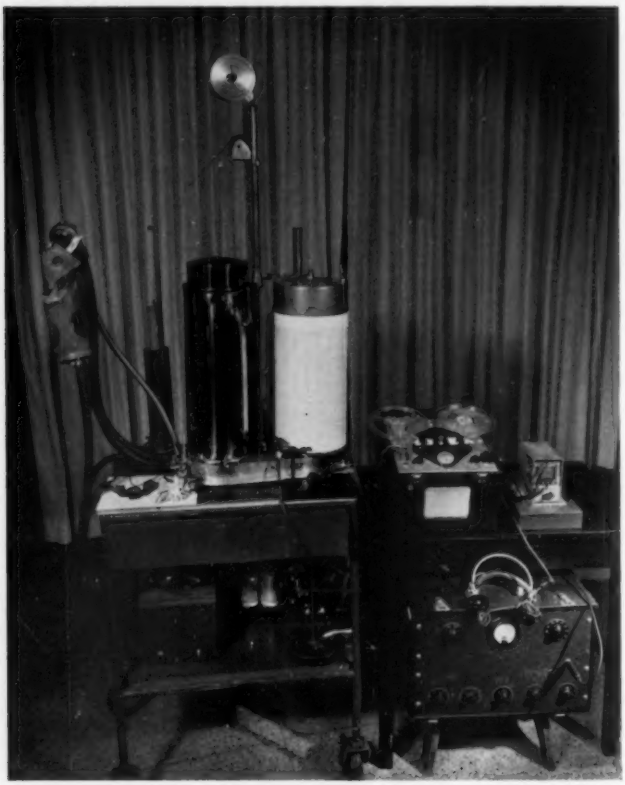

Fig. 2. Photograph of apparatus shown diagrammatically in Fig. 1.

The subject (1) was seated next to the equipment. A lip microphone (2) was placed over the subject's upper lip and suspended from wires which looped over the ears. A civil defense-type gas mask (3), covering the entire face, was fitted on the subject. The mask was made airtight, with the addition of sealed foam rubber strips around the edges, and with

the careful adjustment of the straps around the head. The cartridge had previously been removed from the front of the mask and replaced with a metal diaphragm and connection for ¾-inch rubber tubing (4), which led from the opening to the water-sealed oral spirometer (5). This was a specially built simple spirometer with a displacement of 1 cc. per mm. Its purpose was to record the volume of air the subject used in speaking.

A laryngectomy or tracheotomy tube (6) of the correct size for the individual subject was placed in the tracheostomy, with a cushion of sealed foam rubber between the skin and outer rim of the tube, to insure an airtight connection. For the patient who did not use a tube, a special contact tube with sealed sponge rubber pad was placed over the tracheostomy. A metabolism mouthpiece (7) with the flat part removed was slipped over a rubber stopper on the laryngectomy, tracheotomy or contact tube, making it possible to connect a length of rubber tubing (8) from the tube to the large spirometer. This was a Benedict-Roth 9-liter spirometer with extra large size valves and soda lime absorber.

The customary speed of the paper drive mechanism was modified by the addition of a synchronous motor so that the paper moved at a speed of approximately 25 mm. per second.

As the drum revolved, four ink-writing pens recorded the data desired. The pen connected with the large spirometer recorded pulmonary respiration; the pen connected with the oral spirometer recorded the volume of air used in the production of speech; the third pen recorded the speech signal produced by the subject and transmitted through the following route: microphone, high-fidelity audio-amplifier, syllable pulse relay, and telegraph receiver; the fourth pen recorded a signal which was operated manually to indicate when words or phrases were given to the subject and when the subject spoke. The fourth pen was also attached to a timing circuit on the synchronous motor.

When the equipment was ready, the subject was brought into the room, and the purposes of the procedure in which he was about to participate were explained. The lip microphone,

face mask, and tube connecting the tracheostomy with the large spirometer were fitted and adjusted and all connections tested for air leakage.

The recording of the speech and respiratory data for each subject consisted of four sections. During Section I, the subject was asked to breathe quietly without speaking. Only the

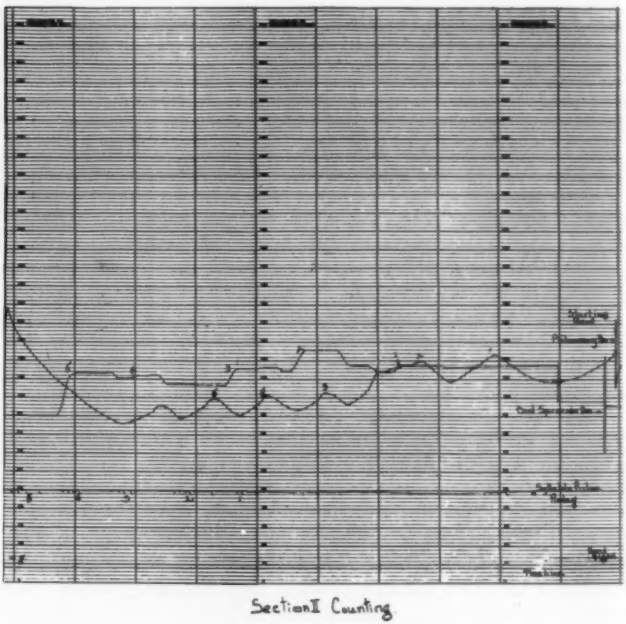

Fig. 3. Representative recording of speech and respiration data (Section II, Counting one to five).

pulmonary spirometer pen functioned during this period. For Section II, the subject was asked to count from one to five at his usual rate and volume (see Fig. 3). In Section III, the subject was asked to repeat five words from a standard "phonetically balanced" list,[2] while in Section IV, he was asked to repeat each of eight five-syllable phrases which were equated

as to time and intensity.[5] During the run, appropriate marks were put on the paper to identify the material. All sounds produced by the subject were recorded on magnetic tape. Time signals, at one-half second intervals, were also recorded on the paper, on a repeat run.

The procedure in the analysis of each completed record was as follows: *1.* A gross inspection was made of the four sections of the record. *2.* The volume of air taken in during the inspiration phase and the volume exhaled during the expiration phase of each cycle of pulmonary respiration were measured in cc. for each of the four sections. To determine these volumes, it was necessary to measure the number of millimeters for each phase of the cycle and to multiply the number by 20.73, which was the volume in cc. of each mm. of vertical movement of the pulmonary spirometer. *3.* The volume of air taken into the mouth for speech and the volume of air used in each speech unit were also measured in cc. This was done by computing the number of millimeters for each phase of the cycle. Each millimeter recorded by the oral spirometer pen represented the movement of 1 cc. of air in that system. *4.* Careful study and comparison of the oral and pulmonary curves were made to determine how many occurred synchronously and how many did not. It should be noted that limited space made it necessary to offset the pens so that comparable moments are not on any vertical line. A transparent glass plate with appropriate markings which showed simultaneous instants on the several curves were used to read the tracings.

Part II: The second procedure involved the use of motion picture photography and speech recordings. The same number of subjects (23) participated.

The following preparations were made for each subject: *1.* the tracheostomy and the area around it were uncovered so as to be clearly visible; *2.* a square of single thickness facial tissue slightly larger than the tracheostomy was secured along its top edge with Scotch tape, which was then attached to the neck just above the tracheostomy in such a way that the tissue would be moved readily by the respiratory air. This delicate curtain provided a visible and sensitive means for ob-

serving the air movement during inspiration and expiration; *3.* the subject was then requested to read the same words and sentences which had been used in Part I, followed by two

1. Quiet Breathing. 2. Intake of Air.

3. Start of Phonation with Raising of Flap. 4. Phonation: Flap blown out.

Fig. 4. Prints made from individual frames of 16 mm. moving picture film giving visual evidence of relation between phonic and pulmonary respiration.

short paragraphs. These paragraphs were constructed to include the sounds which were found by Anderson[1] to be the most difficult for the laryngectomized speaker to produce. A film and sound recording were made as the subject read. Color

film was used throughout and all films were made at sound speed; *i.e.*, 24 frames per second (see Fig. 4).

The visual and auditory data provided by the films and recordings were analyzed to determine the relation between pulmonary respiration and speaking for each subject. Every film was studied frame by frame in a viewer for close observation of the correlation between the stages of lip movement during speech production and movement of the tracheostomy covering. The film was then projected at sound speed as the tape recording of the subject's speech was played, in order to study the relation between the audible production of the sound and evidence of movement of the tracheostomy covering.

RESULTS.

Part I: Analysis of the data regarding volume of pulmonary air revealed that considerable variation in range of volume from section to section was characteristic of the records of the 23 subjects. Wide variation in pulmonary volume from speaker to speaker was also observed when individual records were studied and compared; furthermore, variation of pulmonary volume was generally found to be as great within the sections of an individual record as was the extent of variation in the group as a whole. The nature of this variation made it impossible to conclude whether a relationship exists between speech fluency and the volume of pulmonary air used for respiration by the laryngectomized speaker.

The pulmonary respiration records were of particular interest because they resulted from the use of equipment and procedures different from those employed in previous investigations concerning the relation between pulmonary and phonic respiration and the volume of pulmonary air involved during speech. For the most part, such investigations had to depend on pneumographs which fastened about the chest and abdominal areas. The respiratory apparatus made available for the present study undoubtedly provided a more sensitive instrument as well as a more direct means of obtaining the desired information.

Part II: Analysis of the moving picture and sound record-

ings confirmed the results of the respiratory tracings in regard to the coordination of pulmonary and phonic respiration. The two occurred synchronously in all of the subjects.

The results of this section of the study support Stetson's observation that "records of excellent esophageal speech show that the subject breathes just as in normal speech."[16]

SUMMARY.

Twenty-three laryngectomized speakers served as subjects for respiratory tracings and moving picture photography, to provide data to determine whether pulmonary respiration is significantly synchronous or asynchronous with the oral inspiration and expiration of air for esophageal voice. Volume of air used in the inspiration and expiration phase respectively of every cycle of pulmonary and phonic respiration were also computed.

Positive synchrony between pulmonary and phonic respiration was demonstrated in each of the 23 subjects.

It is apparent from these studies that there is a wide variation in the volume of pulmonary air and oral air in different patients. There is no correlation between *1.* the volume of pulmonary air and the volume of oral air in any individual patient; *2.* between speech fluency and volume of pulmonary air; or *3.* between speech fluency and volume of oral air.

It was not possible to determine accurately from the sound recordings whether or not there was any relationship between loudness of the voice and volume of either pulmonary or oral air.

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School of Speech, Northwestern University, Evanston, Ill.

HOARSENESS IN LARYNGEAL PATHOLOGY. A REVIEW OF THE LITERATURE.

JOHN M. PALMER, Ph.D.,

Seattle, Wash.

INTRODUCTION.

Three pathologic conditions, among others, frequently result in the vocal condition known as hoarseness. These three are laryngeal carcinoma, paralysis and tuberculosis. It is the intent of this paper to present a review of the pertinent literature relating to the symptom in each of these etiologic categories. Recent years have produced little on hoarseness, and this writer intends to re-present the subject of hoarseness for serious consideration by all specialists.

Hoarseness is a symptom of laryngeal pathology. It may well be symptomatic of other conditions, but a basic tenet held by otolaryngologists and speech therapists is that this symptom is a danger signal of the larynx.[12] An extreme view of this is represented by Jackson and Jackson: "The chief symptom of laryngeal disease is hoarseness; it is absent only when the cords and the motor mechanism are entirely free from disease."[11]

The exact nature of hoarseness has been a matter of debate and discussion, not so much from the point of view of origin or therapy, but more from the problem of defining and describing it. The answers have centered around acoustical terminology, physiological, neurological, mechanical and psychological. In spite of what appear to be extreme difficulties inherent in solving this matter of definition and description, and because of the deliberate attempt here to focus attention on pathology, it is my intent to treat hoarseness as do medical pathologists and practitioners, and to utilize their definitions and descriptions.

Description. Jackson and Jackson[11] state that hoarseness is a general term applied to a rough quality of voice. Frank's[4] definition has gained acceptance because it is somewhat more

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specific: hoarseness is "any alteration in the speaking voice which results in a roughness or rasping character in the voice." Speech therapists and acousticians would add to these in detail, perhaps not in spirit. Every laryngologist has heard hoarseness in speech, and though he might not desire to define or describe it, it seems sure that he would certainly recognize it.

In demonstrating hoarseness, the laryngeal mechanism has failed partially or totally in one or more of its three major functions. Normal phonation requires that the vocal cords are capable of approximation, tension and vibration. Should any of these movements receive interference, hoarseness may result.[11]

Approximation may be disturbed by a growth between the cords, by feebleness, by paralysis, by interference with the movement of the cricoarytenoid joint, or by cicatricial concave cordal edges.

Tension may be disturbed by paralysis, feebleness, fixation of the joints, thickening of the cord, or by cicatrices of the concave cordal edge. As Jackson and Jackson define tension, it is an elastic recoil reinforced by muscular support of the cords; it is not a stretching like a violin string.[11]

Vibration may be disturbed by thickening of the cordal edges, by a growth acting as a damper, by feeble tension, or by incomplete approximation. Actually, approximation, tension and vibration may all be interfered with by the aggressive, usurpative action of the ventricular bands. "These bands can produce only hoarse phonation which in these cases is substituted for cordal phonation."[11]

Krieger notes that in the inflammatory type of hoarseness, approximation of the cords is only slightly interfered with, but the tension is very much impaired, varying with the severity of the condition present while the vibration varies directly with the tension.[12] In line with this same philosophy of complete investigation, Frank claims that a patient with hoarseness requires a thorough examination, laryngoscopy, indirect or direct when the mirror examination is not satisfactory; an X-ray of the chest, and occasionally of the neck; Was-

sermann; complete blood count and urinalysis. Biopsy should be performed when there is the slightest suspicion of a malignancy.

LARYNGEAL CARCINOMA.

It is evident that laryngeal carcinoma demands immediate consideration. Recent clinical experience has demonstrated that it is curable in a large proportion of cases *if diagnosis is made early*. On the other hand, in situations of late diagnosis, the trend is reversed to almost complete lack of cure. "Of primary importance is the recognition by the lay and the medical public that persistent or intermittent hoarseness in an adult means cancer, until it is proven otherwise by proper diagnostic means."[9]

Laryngeal carcinoma comprises about two per cent of all human malignant cancers. Like cancer of the mouth this disease occurs most often in middle and old age, and predominantly in males. The incidence of intrinsic and extrinsic cancer is about equal.

Specifically, as far as age and sex of affliction is concerned, it has been found in one statistical study that of 500 patients, 92 per cent were males, and that the average male age was 60, while the average female age was 40.[21] It was further noted that in cases of carcinoma of the larynx, with no treatment being received, 50 per cent of the sufferers are dead within one year, and that 95 per cent are dead in two years; yet, with the proper diagnosis and treatment, the five year level is as high as 82 per cent, and possibly higher today.[20]

Etiology. The pre-existence of a chronic local irritation which so often may be shown to play a part in the causation of malignant growths in the oral cavity is not readily demonstrable in the case of cancer of the larynx. Long felt to be etiologic factors in laryngeal cancer are excessive smoking with inhaling, voice strain and syphilis; yet, research has yet to demonstrate a significant etiologic role in most cases. As Martin states, "The disease usually appears to arise spontaneously."[16]

That growths in the laryngeal mechanism are the most com-

mon cause of hoarseness is hardly to be denied. Zinn states that neoplasms have been found in the largest number of cases; further, he reports that the symptom depends upon the involvement of the motor mechanism, on the location of the tumor, and whether or not it interferes with normal laryngeal movements.[26] These movements are, of course, those described earlier: approximation, tension and vibration.

Benign growths of the larynx, too, are responsible for this vocal condition; in fact, some authorities feel that there are many more benign than malignant lesions of the vocal cords, and, after the removal of such a benign tumor, one should expect a nearly normal voice in nearly all cases. Easing the situation somewhat is the fact that benign lesions of the vocal cords usually do not constitute a threat to life. It must be remembered, though, that they do cause disability manifested by hoarseness, and some benign lesions may be forerunners of malignant lesions.[7]

Symptoms. The fact that hoarseness does occur in laryngeal carcinoma is well known. Zinn reports that in 25 cases of total laryngectomy, 20 were hoarse preoperatively (89 per cent); that of another 178 cases, 100 were hoarse; and that of a group of 144 carcinoma victims, 134 (93 per cent) were hoarse.[26] Jackson and Jackson found that of 410 carcinoma cases, 389 (95 per cent) were hoarse.[9]

A growth situated anteriorly in the anterior commissure may so prevent approximation of the cords as to cause complete loss of voice (aphonia); on the other hand, a larger soft growth located below the cords may not interfere with adduction, and give rise only to the symptoms of local irritation. Paroxysmal hoarseness is often observed in such cases, especially after loud talking. A small tumor situated above the cords may give rise to very little trouble and cause no subjective symptoms. "In the great majority of cases, however, hoarseness is the first manifestation," state Lederer and Hollender.[14] Agreeing with this point, Jackson and Jackson note that the importance of location of the growth on the symptom depends upon whether or not the motor mechanism is involved. "By motor mechanism, we mean the cord, the aryte-

noid cartilage, the cricoarytenoid joint or the nerves or muscles concerned in their motility."[9]

It must be stated that the symptom under consideration, *hoarseness*, may not be present while the condition, *carcinoma of the larynx*, may well be. It is, however, with the symptom that this study is concerned; and it must be remembered that one of the earliest, most important and most common symptom is that of hoarseness. The absence of hoarseness in laryngeal cancer usually occurs when the growth is located above the glottis anteriorly. The symptoms for an advanced supraglottal carcinoma are frequently phonatory:: "The voice becomes more and more affected as the growth increases in size and eventually it becomes a wheezing, rasping, almost aphonic voice that is quite suggestive to those who have heard it often."[10]

Vocal cord cancer is evident in practically all cases first by hoarseness. The progress of the disease is usually slow, and the hoarseness may persist as the only symptom for as long as one to three years in untreated patients before the airway is sufficiently diminished to produce dyspnea. As the disease infiltrates deeply into the vocal cords, complete aphonia results. If, on the other hand, the lesions tend to fungate rather than infiltrate, husky phonation may be accompanied by gradually increasing dyspnea as the aperture of the glottis is narrowed.

Diagnosis. Inasmuch as hoarseness can be the symptom of a mortal illness, this fact and the observation of a new growth on the vocal cord should lead to a diagnosis that will either prove or disprove a cancerous condition.[8] Lederer and Hollender claim that "A conclusive diagnosis involves a careful survey not only of the larynx and its environment, but also of the body as a whole. A malignant tumor occasionally resembles chronic hypertrophic laryngitis, or a benign fibroma."[14]

Laryngeal carcinoma actually has three symptoms: persistent pain and discomfort on swallowing, the appearance of cervical metastasis in cancer of the extrinsic larynx, and hoarseness in intrinsic larynx (vocal cords) involvement.[16] Obviously, hoarseness, although insidious in growth, remains the most outstanding symptom; Jackson and Jackson place

their hope for accurate diagnosis on this symptom alone, feeling that it is the one symptom for a number of months, perhaps a year.[9]

Because the vocal cords are the site of most cancers, it appears evident that motility in phonatory and respiratory activities plays an important role in the development of the tumor. It is not known definitely what causes the malignancy, as was stated previously, although Jackson and Jackson state, "The location of 85 per cent of cancers on the most abused part of the larynx, the middle third of the cord, renders it logical, even if not probable, that vocal abuse is one cause of cancer of the larynx."[9]

Summary. It appears, then, that cancer of the larynx seldom occurs without hoarseness. Hoarseness, especially of the persistent type, is obviously a danger signal; however, there can be a cancerous growth within the larynx without hoarseness. In the main, though, hoarseness is the first sign of cancer, and should be the signal to the speech therapist to refer the case to medical practitioners for complete laryngoscopic and other examination.

LARYNGEAL PARALYSIS.

Introduction: Neurological disorders of the larynx generally result in malfunctioning of the musculature controlling phonation. If by paralysis or paresis, these muscles are interfered with partially or completely, the symptom of hoarseness, as well as other vocal abnormalities familiar to the practicing speech therapist, may appear.

Etiology. In general, in paralysis of the larynx, the voice may be hoarse, and the patient may not be able to speak above a whisper, and there is special difficulty with vowel sounds. A similar change occurs in laryngitis and in tumors of the larynx. In a unilateral laryngeal palsy, or in recurrent paralysis, the voice is low-pitched and hoarse, but occasionally there is almost complete unilateral paralysis without appreciable effect on phonation, due to unimpaired adduction and to crossing of the midline by the normal cord. In bilateral abductor paralysis speech is moderately affected, but in bilateral total palsy, it is lost. "In paralysis of the crico-thyroid, which

is supplied by the superior laryngeal nerve, the voice is hoarse and deep and fatigues quickly. In adductor palsy, usually psychogenic, the loss of speech is out of proportion to the involvement."[2]

The description of paralysis of the larynx can be limited to two aspects: the opening and the closing of the cords. In this respect, Jackson and Jackson offer simple definitions. They term abductor paralysis as failure of the glottis to open from lack of impulses normally transmitted by the recurrent laryngeal nerve.[10] Similarly they describe adductor paralysis of the larynx as an impairment of closing movements due to impaired innervation.[11]

Friedman summarizes the general belief, briefly stating that in all progressive organic lesions of the centers and trunks of the motor laryngeal nerves, the fibers supplying the abductors of the vocal cords become involved much earlier than the adductors.[5] According to Sjoberg,[22] there is some significance in the bilaterally symmetrical innervation of the larynx. In supranuclear lesions, *i. e.*, above the nucleus ambiguus, paralysis of the larynx can occur only when the lesions are so great that double-sided interruption of the corticobulbar fibers takes place. The paralysis is then bilateral and of the spastic type. On the other hand, unilateral paresis of the vocal cord can never occur through supranuclear lesions.

Sjoberg describes the paralysis of the vocal cords as due to interruption of the peripheral conduction as a result of injury to the recurrent and vagus nerves in the neck or cavity of the thorax.[22] As an example of the neurological involvement of the larynx due to activities in surrounding areas, Froeschels points out that until recently more than 50 per cent of the cases showed paresis or palsies in the region of the recurrent nerve after operations on the struma; however, this number has been remarkably reduced by modern surgical technique and by research in dissection of the distribution of the recurrent laryngeal nerve.

Some 85 years ago William Marcet attributed another influence to the development of paralysis of the larynx, saying that paralysis or weakness of the laryngeal muscles causing loss of voice very frequently attends hysteria.[15] This view has

numerous supporters today and must not be rejected by the speech therapist.

Lederer has described neural pathologies in a convenient fashion:

> Organic paralyses of the larynx fall into four clinical groups: neuropathic, encephalopathic, myopathic, and a combined form of myopathic and neuropathic types. When the paralysis is neurogenic in form, there is absence of local pathologic change. The myopathic form is characterized by the presence of some local lesion, such as swelling, tumor, or ulceration. A great deal of evidence has been presented to support Semon's "law" (that the abductor muscles—cricoarytenoid— of the larynx show a greater proclivity to motor impairment than the adductors—thyroarytenoid, lateral cricoarytenoid, interarytenoid, transverse and oblique). Some laryngologists, however, believe that there are other mechanisms involved; undoubtedly other nerves or muscles keep the vocal cord or cords adducted despite the destruction of the major nerve supply (recurrent laryngeal).

In discussing the nature of abductor paralysis, Berry and Eisenson report that the most common accident is the severing of the recurrent laryngeal nerve in thyroidectomy, as did Froeschels.[6] They further state that less frequent is a paralysis of this nerve through pressure of a goiter, or aneurysm of the aorta. The circuitous routing of this nerve accounts for the major difficulties. Since this nerve furnishes the motor innervation to the posterior cricoarytenoid muscles of the larynx, any interruption of impulses will result in a failure of the glottis to open (abductor paralysis of the larynx).[1] Another supporting view of the importance of the recurrent laryngeal nerve is presented by DeJong, who states that the most common type of laryngeal palsy is the result of unilateral recurrent nerve lesion.[2]

A simple but complete description of the neurophysiology of adductor palsy has been given by Orton. He emphasizes that the cricothyroid muscle has a marked adductor function. This muscle is responsible for maintaining the cord in the so-called median or para-median position following complete recurrent laryngeal nerve paralysis. Experimental evidence and visual observation indicate that following section of both recurrent laryngeal nerves, elimination of the function of the external branch of the superior laryngeal nerve produces a marked abduction of the vocal cords, increasing the laryngeal airway.[19]

When the paralysis is confined to the adductors of the vocal cords, according to Friedman, it is almost invariably bilateral and is due to functional disturbances in the central nervous system. The organic lesions produce either respiratory difficulty alone, or both respiratory and phonatory difficulties. A partial paralysis of phonation has been observed in multiple sclerosis and bulbar paralysis. Lead poisoning has been known to produce paralysis of the abductors as well as unilateral complete recurrent palsy.[5]

Three stages have been described in recurrent paralysis: DeJong notes that the first manifestation is lessened abduction on the involved side, due to isolated involvement of the posterior cricoarytenoid; next, there is tension, of secondary contracture of the adductors: and finally there is complete paralysis with the cord in the cadaveric position The uninvolved cord crosses the midline.[2] Maxwell sees it somewhat differently in respect to the eventual position of the cords in this condition when he reports that the paralyzed vocal cords come eventually to lie in the midline as a result of recurrent nerve injury.[17]

Another neuropathology of the larynx is concerned with tensor, or thyroarytenoid paralysis. It is simply defined by Jackson and Jackson as failure of the tensor mechanism due to lack of motor impulses.[10] These authors state that as an isolated affection tensor paralysis properly so-called is rare; yet, as a result of total cadaveric paralysis, it is common.

Symptoms. In the case of abductor paralysis of the larynx where the glottis fails to open, Berry and Eisenson describe the result as a completely voiceless condition of the patient, and the folds are abducted into the cadaveric position. Gradually, as the cords approach the midline, he is able to phonate, but does so with air waste and husky quality to the sound. Breathing is stridorous. If the paralysis is limited to one cord, the normal cord will move back beyond the normal excursion in an effort to compensate for the immobile cord which remains in the midline; the total effect on the voice is practically the same.[1]

In general, DeJong agrees with this description, but uses

the term "hoarseness" in describing the effect of the palsy. He has observed that in unilateral abductor palsy the involved cord lies close to the midline and cannot be abducted on inspiration. Too, in his opinion, the voice may become hoarse, but in general phonation and coughing are but little affected, since adduction is normal. In bilateral abductor palsy, both cords lie close to the midline and cannot be abducted. DeJong notes that the voice may be hoarse; but phonation is often only slightly affected, because both cords can still be adducted, and coughing is normal.[2]

Hoarseness plays a part in the description of bilateral abductor paralysis given by Jackson and Jackson, for they have noted that the outstanding characteristic symptoms of bilateral recurrent laryngeal paralysis are dyspnea and stridorous breathing; the voice may be little or not at all affected, but it will be noted that the phonation must be interrupted for a stridorous inspiration at frequent intervals. In complete paralysis with a cadaveric position of the cords, according to these authorities, there is a wheezing sound to the cough, and the voice is a very hoarse stage whisper without true phonation.[10]

Strother, too, notes that hoarseness is not a symptom of this type of paralysis. By his description, when bilateral paralysis follows the more common course, there is an initial aphonia during which the cords are abducted in the cadaveric position. Gradually, the voice is recovered, but as the voice returns, due to changes of the cords toward the midline position, the glottis becomes narrower and respiration becomes increasingly difficult. He notes, however, that bilateral paralysis cases present themselves to the speech therapist much less frequently than do cases of unilateral paralysis.[24]

Jackson and Jackson agree with Strother that unilateral abductor paralysis is much more common than the bilateral variety. In addition, they find the lost motion occurs more frequently on the left side than the right and that unilateral paralysis is usually peripheral, due to lesions in the neck or the upper part of the thorax;[10] and with one cord paralyzed the patient may phonate perfectly but it will usually be discovered that there was a period when he was hoarse.[10]

It seems that with abductor paralysis, the symptom of hoarseness has not been listed as a common one; yet, there are definite changes in the ability to phonate normally, changes which the speech therapist should recognize.

Hoarseness, however, has definitely been attributed to conditions of adductor paralysis of the larynx. The chief symptom in unilateral adductor paralysis, according to Jackson and Jackson, is hoarseness; a wheezy husky voice accompanied by air waste on attempted phonation.[11]

DeJong describes adductor palsy differently, noting that the cords are not adducted in phonation, and the voice is either lost or cannot be raised above a whisper. He notes that the difficulty is almost always bilateral and is generally of psychogenic origin. Unilateral adductor paralysis is occasionally seen in trauma and in peripheral lesions; there is paralysis of one lateral cricoarytenoid with hoarseness and also impairment in coughing.[2] In the case of recurrent paralysis, DeJong summarizes by stating that "the voice may be coarse and husky, and there is loss of the ability to sing."[2]

In a total unilateral palsy, DeJong notes that both adduction and abduction are affected, and that the involved cord lies in the cadaveric position, motionless in misabduction. The voice is low-pitched and hoarse, and there is difficulty in coughing, but phonation may not be seriously affected since the normal cord may cross the midline.[2]

As far as the effect of tensor paralysis, Jackson and Jackson state that the voice is weak and husky with air waste; a little conversational use causes the patient to say his "voice is tired," or his "throat is tired."[10] DeJong describes a slightly different manifestation, observing that in bilateral thyroarytenoid paralysis the glottis has an oval instead of a linear appearance during phonation; the voice is hoarse, but there is neither dyspnea nor stridor. In paralysis of the arytenoid the glottis is closed only anteriorly during phonation, according to DeJong, and inspiration is normal.[2]

Phonation as well as respiration are disturbed in progressive bulbar palsy. In the later stages of the disease, the voice becomes monotonous and feeble. Hoarseness may supervene and

go on to complete aphonia. The cough is weak and hollow. Laryngoscopic examination in the beginning of bulbar palsies may show relatively little, according to Friedman, and later on, paresis of the adductors appears with insufficient closure of the vocal cords so that phonation is difficult.[5]

Summary. In unilateral paralysis, a speech therapist's cooperation is of great value to aid the patient in obtaining maximum results during the time that the cord, which is healthy, crosses the midline as an attempt of the physiologic sensibility of the larynx to compensate for the immobile cord. In recent years, progress has been made by external surgical means to relieve the patient of respiratory embarrassment in cases of bilateral paralysis, according to Lederer. Such procedures immobilize and draw aside an arytenoid, thereby permitting a small but functional airway without influencing the phonatory responses too adversely.[13]

It appears, then, that in instances of abductor paralysis, phonation is not always affected; thus the disorder under study, *hoarseness*, may not be attributed to faulty abduction of the vocal cords; however, the opposite aspect, that of adduction, does seem to present vocal problems, including hoarseness, when its function is disturbed (paralyzed). This is especially true in cases of recurrent nerve paralysis. One additional neurological malfunction concerns the total unilateral paralysis, affecting both adduction and abduction, with a resulting hoarseness in the syndrome.

LARYNGEAL TUBERCULOSIS.

Introduction. Although it is not the most common disease entity resulting in or causing hoarseness, large numbers of individuals infected with pulmonary tuberculosis show symptoms of hoarseness, indicating a spread of the primary disease to the larynx.

By definition, tuberculosis of the larynx is a lesion therein due to *Bacillus tuberculosis*.[10] It is an infectious and contagious disease of the larynx and is secondary to pulmonary tuberculosis. It is very rarely primary and is usually chronic from the onset. It is characterized by infiltration, formation of

tubercles, granulomata, and ulcers in the walls of the larynx, followed later in some patients by edema, perichondritis, chondritis, or even necrosis.[22]

Thomson, writing in England in 1924, stated that there was no specific disease of the larynx so common as tuberculosis.[25] Notwithstanding the differences in geography, economics, and time, current American medical experts claim that tuberculosis is still one of the commonest of laryngeal lesions, excluding acute and chronic laryngitis.[10] It is more frequently met with in individuals between the ages of 20 and 45. It attacks both sexes, but is more common in men than women to the ratio of 3:2. From 3 to 25 per cent of the patients with pulmonary tuberculosis manifest laryngeal tuberculosis.[14,23]

Spencer[23] quotes Dworetzky[3] as having found hoarseness in 82.6 per cent of one of his series of early cases. In his observations of this disease, Thomson came to similar conclusions to those of Ormerod when he found that in 477 patients who had a lesion of the larynx, the most common complaint was some alteration of voice. He noted that change of voice may come so insidiously and cause so little general inconvenience that a patient and his friends may never have observed that a previously clear and musical voice had become toneless, rough, harsh, or even hoarse.[25]

Etiology. The involvement of laryngeal tuberculosis is a progressive process, according to Thomson,[25] with the posterior commissure (arytenoids, interarytenoid region, and vocal processes) most commonly affected. Following this, the vocal cords become involved. Next in order comes the anterior commissure. Last is the epiglottis in the progressive nature of the disease.

It seems clear that the somewhat complicated mechanism of approximation and vibration, as well as tension, of the vocal cords can be deranged in many ways, any one of which would result in some degree of hoarseness. The approximation of the cords can be interfered with by irregularity in the edges of the vocal cords themselves, either nodular or edematous, or by the interposition of hypertrophic tissue from the interarytenoid space between the vocal processes of the aryte-

noids. Cord approximation can also be affected adversely by interference with the approximation or rotation of the arytenoid cartilages. This can be caused by lesions which interfere with the fibers of the recurrent laryngeal nerve. Such lesions occur as a fibrosis at the apex of the lung, pressure from enlarged glands, or infiltration at the penetration of the muscle by the nerve.

Rotation of the arytenoid cartilages can be disrupted by any of the above conditions as a result of laryngeal tuberculosis, as well as by tuberculosis infiltration in the region of the cricoarytenoid joint. This can occur with or without implication of the joint or the cartilage, and can affect the rotation of the arytenoids in such a way as to interfere with approximation or vibration.

The vibration of the cords may be disturbed in a number of ways. Paralysis of the internal tensor muscles by lesions affecting the recurrent nerve, atony of these muscles by tuberculous debilitation of the patient, or infiltration of the muscle by tubercles have the effect of abolishing the tone of the vocal cord, thus affecting the tension and the vibration activities. The cord remains bowed on attempting to phonate and the voice is a very weak sound, particularly when the condition is bilateral. Infiltration of the vocal cord, with or without edema, prevents proper vibration as well as interfering with approximation.

The role of the ventricular bands is no small one in cases of laryngeal tuberculosis. Swelling of these bands, protrusion of swollen and infected membranes from the mouths of the ventricle of Morgagni and subglottic infiltration have the effect of dampers of the vocal cords and again prevent proper vibration.

In brief, then, hoarseness is produced by any lesion, however small, of the vocal cords, and by any but the very smallest of lesions in the ventricular bands or arytenoids, according to Ormerod.[18] He does state, however, that lesions restricted to the epiglottis or even to the aryepiglottic fold do not produce hoarseness.

Symptoms. Because it is secondary to the pulmonary condition, laryngeal tuberculosis is late in developing, notes Zinn,[26] and exhibits varied symptoms. Painful swallowing exists even in the absence of ulceration. Lederer and Hollender note that difficulty in swallowing is common among those patients in the presence of marked ulceration and infiltration, especially of the epiglottis or aryepiglottic folds. Change in the voice is at first absent; however, hoarseness may follow a paroxysm of coughing or an effort at speaking. Alteration of the voice, say these authors, depends upon the degree of involvement of the phonatory apparatus.[14]

Thus, authorities are fairly well agreed that the symptoms of tuberculosis are, in the main, threefold: hoarseness, pain and dyspnea, occurring in the order of frequency. Ormerod[18] reports that hoarseness was noted 290 times in 300 cases in which the symptoms and the data had been carefully analyzed. He states that in the slighter degrees the variation is very noticeable. A patient who has an obviously husky voice one day may have a normal voice the next, and hoarse again two or three days later. Some individuals with this disease entity report they are hoarse on waking and improve during the day; others have the opposite experience, waking with a good voice which apparently tires during the day.

According to this same writer, Ormerod, the hoarseness of tuberculosis of the larynx is never raucous, as in a benign or malignant tumor of the cord. The voice may be bitonal or somewhat rough, but there is always a sensation of moistness about it, and as the hoarseness increases the impression is made of collected mucous in the larynx. This authority states that the voice weakens, and eventually there is nothing left but a moist whisper.

Spencer[23] agrees generally in the progressive nature of the symptoms. He found that the subjective symptoms often begin with slight hoarseness. This could be present only in the mornings after the patient arises, or after a change in position if the patient is kept in bed, or after a paroxysm of coughing.

Jackson and Jackson stress the fact that voice is weak, not

just impaired in quality—often weak out of all proportion to the lesion visible in the mirror.[10] According to them, this is due to a myasthenia affecting the thyroarytenoid muscle, which is usually the first muscle to show the weakening that later affects all muscles of tuberculosis patients. This muscular weakness may produce hoarseness, even aphonia, before there is any tuberculosis lesion in the larynx. Usually, state Jackson and Jackson, the voice deteriorates until it becomes a rough whisper, as though somewhat forced. In the earlier stages the cords may approximate very well and their edges may look thin enough to vibrate, but they seem to be inefficient in the production of sound.

If the patient is not kept silent, talking often precedes the hoarseness, according to Spencer. Then, later in the disease, the hoarseness becomes constant and soon results in aphonia. Jackson and Jackson, too, strongly support the efficacy of a silence regimen. They seem to believe that the prognosis in the case of a patient who will not and cannot stop talking is bad; it is next to impossible for a patient who talks all the time to get well of a laryngeal tuberculosis. These authors also state that the same may be said for those patients who indulge in alcohol and tobacco.

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THE LOW COLLAR INCISION FOR WIDEFIELD LARYNGECTOMY.*

WALTER P. WORK, M.D.,
San Francisco, Calif.

INTRODUCTION.

Since Kocher[1] first popularized the low collar incision, it has been used by surgeons throughout the world for thyroid surgery as well as other surgical conditions in the neck. This incision has long been recognized as the one which will give satisfactory cosmetic results and which will allow adequate exposure of the surgical field.

The low collar incision was used by Col. Wendel Weller and the author at Letterman General Hospital in 1948 for widefield laryngectomy in a patient with squamous cell carcinoma of the larynx. Since that time the author has used this incision successfully in patients who require widefield laryngectomy and in those who require primary block dissection combined with widefield laryngectomy. When primary block dissection is combined with widefield laryngectomy, an extension of one end of the incision is carried upward over the sternocleidomastoid muscle to the mastoid process. In reality, such an upward extension of one end of the incision allows satisfactory exposure of the surgical field, and is J-shaped on the left side of the neck and reversed on the right side, as contrasted with the Glück-Soerensen incision which is U-shaped.

It is of further interest to point out that some years ago Babcock[2] used a high collar incision for laryngectomy, excising a lower round piece of skin and subcutaneous tissue for creation of the tracheostomy.

It is the purpose of this paper to record our experiences with the use of the low collar incision for widefield laryngectomy in patients with squamous cell carcinoma of the larynx who have been treated during the past eight years.

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* From the Otorhinolaryngology Departments of the University of Stanford Medical School, St. Luke's Hospital, and Fort Miley Veterans Hospital.

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ADVANTAGES.

1. Exposure of the surgical field is adequate. The upper flap can be dissected well above the hyoid bone, and the lower flap can be dissected to the suprasternal notch. Such wide exposure allows removal of the hyoid bone above, adequate handling of the strap muscles and thyroid gland lobes and isthmus, and removal of as many tracheal rings as necessary with the specimen. Once the larynx is removed and the closure of the pharynx and esophagus is accomplished, suturing of the flaps and creation of the tracheal stoma are readily done.

2. Cosmetically the low collar incision has the advantage of being placed below the collar line of the average male so that the scar and stoma are covered by the shirt collar. In the female, an appropriate necklace can be worn to cover this area.

3. For the most part the tracheal stoma which is created is round and symmetrical. In over 97 per cent of the cases, no cannula of any type need be worn by the patient postoperatively.

4. As pointed out above, primary block dissection can be done simultaneously with widefield laryngectomy by the upward extension of one end of the incision. Further, secondary block dissection can be done, using the conventional T-incision without jeopardizing the blood supply of the original flaps.

5. If a sinus tract or fistulous opening develops postoperatively in the area of the incision, drainage in most instances is toward the supraclavicular spaces (ends of the incision) rather than toward the tracheal stoma area.

6. The development of esophageal voice by the patient proceeds in the normal manner.

7. It will be found that the fascial layers beneath the platysma muscles are relatively bloodless during the creation of the flaps.

DISADVANTAGES.

1. The creation of a relatively large upper flap and the smaller lower flap is time consuming.

2. A higher percentage of postoperative sinus and fistulous tract formation with neck suppuration will develop in patients when the low collar incision is used, if one does not pay particular attention to meticulous postoperative treatment (as will be pointed out later).

3. This incision is not suitable for all patients who require widefield laryngectomy. Patients with gross X-ray changes of

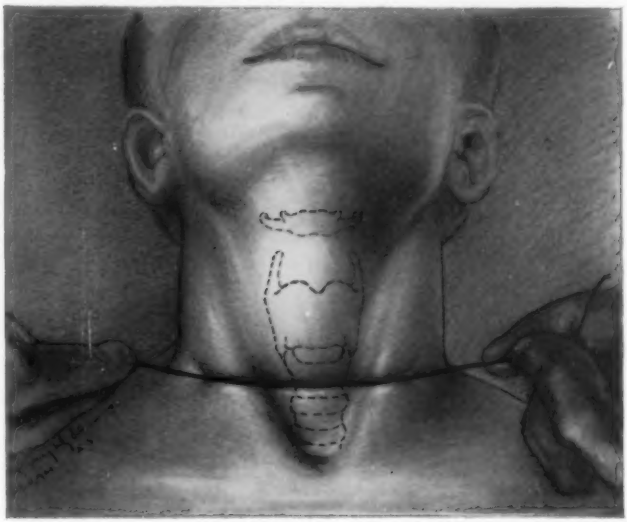

Fig. 1. Low collar incision is being outlined by suture material.

the skin such as atrophy, loss of blood supply and telangiectasia following irradiation for carcinoma of the larynx should not have a widefield laryngectomy through a low collar incision since the flaps may slough. In this instance a T-incision is recommended. Experience has shown that patients who have recently been irradiated usually do not show skin changes which would contraindicate the use of the low collar incision.

TECHNIQUE.

In performing widefield laryngectomy, the low collar incision is outlined by suture material between the lower border of the cricoid cartilage and the first tracheal ring, as shown (see Fig. 1). In most cases this approximates a distance of two finger breadths above the suprasternal notch. By thus marking the skin, the incision can be made symmetrical in relation to the surface contours of the neck. The surgeon

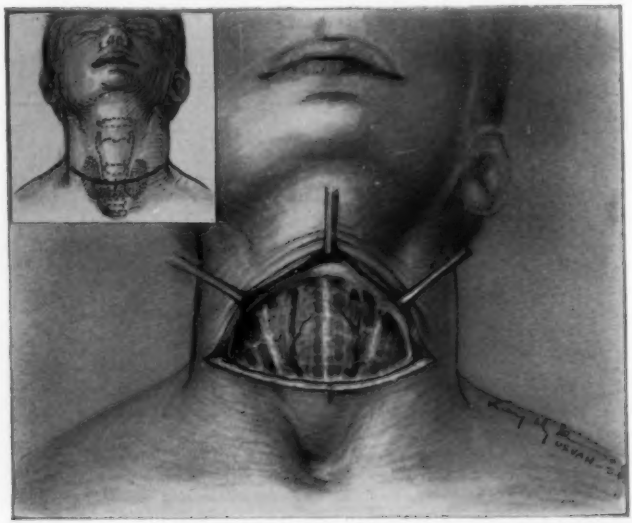

Fig. 2. Insert shows vertical midline marking plus low collar incision outline. The upper flap is being created.

can vary the outline of the incision as he desires in relation to the constant anatomical landmarks, namely, the cricoid cartilage and the first tracheal ring.

After the skin has been marked with suture material, a small vertical mark is made in the skin directly over the midline of the neck so that this mark can be used at a later stage in the operation to help approximate the upper and lower skin flaps to the severed trachea in both a dorsal and ventral position, respectively (see Fig. 2). The incision is carried through

the platysma muscles, which are then lifted with the skin flaps thus exposing the superficial layer of the deep cervical fascia and venous channels (see Fig. 2).

The upper and lower flaps are completed, as illustrated in Fig. 3, with the cervical fascia, venous channels and muscles in view. The suprahyoid muscles and their fascia are exposed, and this allows complete removal of the whole hyoid bone with the larynx. Laterally, the bellies of the sternocleidomastoid

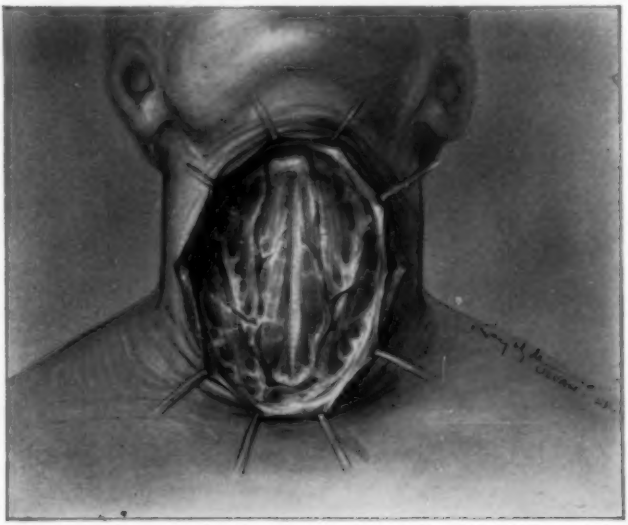

Fig. 3. Upper and lower flap created showing adequate exposure of the surgical field.

muscles are exposed. The inferior flap is elevated to the suprasternal notch, assuring adequate mobilization for closure. Complete mobilization of the lower flap aids closure, particularly in those patients with enlarged thyroid glands and in those cases where several rings of the trachea have been removed with the specimen.

The widefield laryngectomy from this point on proceeds in a step-by-step manner and Fig. 4 shows the neck after the

larynx and adjacent structures have been removed; in fact, closure of the pharynx and peripharyngeal muscles has been completed in a T-shaped manner. The severed strap muscles have retracted beneath the sternocleidomastoid muscle bellies and for that reason are not illustrated. The cut isthmus of the thyroid gland has pulled upward permitting the severed trachea to assume a more ventral position. Note that the rub-

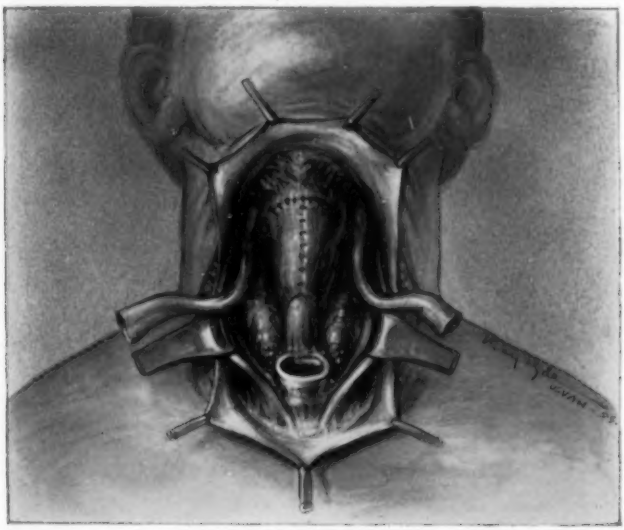

Fig. 4. The larynx and adjacent structures have been removed and closure of the pharynx completed.

ber drains are placed high in the gutters of the wound, and are positioned laterally throughout. By placing the drains thus, the vulnerable T-closure area of the pharynx is allowed to be in apposition to the under surface of the flap from the time it is placed for closure.

After hemostasis is secured, and the drains have been placed, closure of the wound is completed. The formerly marked area of the midline of the upper flap is sutured to the

dorsal midline of the trachea (see Fig. 5, trachealis muscle area). Even though an intratracheal breathing tube is in place, suturing is done about the tracheal stump. Skin is approximated to the tracheal mucosa at all times; and, laterally, tracheal cartilage is included in some of the individual interrupted sutures. These sutures are so placed that each includes a generous amount of upper flap skin so that there is an ac-

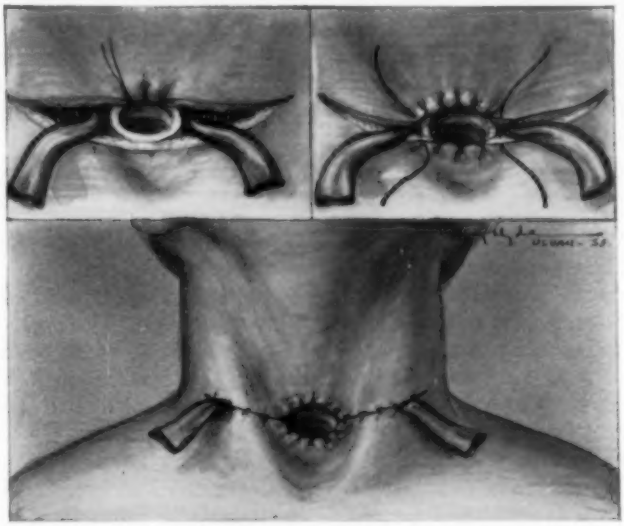

Fig. 5. Steps in closure of the low collar incision are illustrated.

tual puckering of the skin about the tracheal stump. In healing this allows equal tension and pull of scar tissue about the stoma. The upper flap is used only to encircle the dorsal one-half of the severed trachea.

In a like manner, the lower flap is used to encircle the ventral half of the trachea, again starting by suturing the mid-portion of the incision to the midline of the ventral area of the trachea. The same type of puckering interrupted sutures is used. At the sides of the stoma, the two flaps meet and are

anchored by interrupted sutures. The drains are pushed to the lateral margins of the incision and sutured in place. The remaining bilateral portions of the incision are closed with deep interrupted catgut sutures (platysma muscle and fascia approximation), and the skin is closed with interrupted black silk sutures.

A No. 9 or 10 tracheotomy tube is then placed in the trachea after the anesthetic intratracheal tube has been removed. This tube is anchored firmly about the lower portion of the neck by hernia tapes. A large pressure dressing is then applied. This dressing should be applied so that all movement of the head and neck is restricted. This step in the procedure is considered extremely important since immobilization is one of the main factors in successful healing.

POSTOPERATIVE CARE.

Postoperative care of patients who have undergone widefield laryngectomy through the low collar incision must be meticulous in order to minimize the formation of sinus and fistulous tracts with accompanying suppuration. The pressure dressing is maintained continuously for at least one week, and is changed and reapplied as necessary during this time. The rubber drains are partially removed starting on the fifth postoperative day and are usually completely removed by the seventh postoperative day. Sutures are removed between the seventh and tenth postoperative days. On the average, feeding by tube is continued for ten days. It may be pointed out that at operation the feeding tube is placed in the lower esophagus, and not the stomach, to avoid postoperative epigastric distress. The usual supportive care plus humidification of the atmosphere and the use of antibiotics is continued postoperatively. When the flaps have healed solidly, on an average of four to six weeks postoperatively, esophageal voice instruction is begun. Usually at this time also the metal cannula can be removed permanently.

SUMMARY.

During the past eight years, experience has shown that the lower collar incision can be used successfully for widefield

laryngectomy in the treatment of patients with cancer of the larynx. Further experience has shown that its modification (extension of one end of the incision upward) can be used for widefield laryngectomy with primary block dissection. Advantages in its use both to the patient and laryngeal surgeon far outweigh its disadvantages.

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ANNOUNCEMENT.

The Mount Sinai Hospital, New York, in affiliation with Columbia University announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty given by Irving B. Goldman, M.D., and staff, July 14, 1956, to July 28, 1956. Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

THE RECOGNITION AND SURGICAL TREATMENT OF CONGENITAL OSSICULAR DEFECTS.*†

ROBERT HENNER, M.D.,

and

RICHARD A. BUCKINGHAM, M.D.,

Chicago, Ill.

Minor congenital anomalies of the middle ear may cause significant conductive hearing losses and favor the coexistence of middle ear and mastoid disease. The purpose of this presentation is to relate some of our experiences in diagnosing and treating such deformities.

Hearing rehabilitative surgical procedures can be performed in many patients suffering from congenital defects of the middle ear and external canal by using the advanced techniques of temporal bone surgery which were primarily developed for labyrinth fenestration in otosclerosis.

We wish to present four cases of minor congenital aural defects with conductive hearing loss. Three of these cases were proven at surgery to have a synostosis of the incus and malleus as the chief cause of the hearing loss. Before detailed discussion of these cases, we would like to present a working classification of congenital ear defects that has been valuable to us in deciding the indications and type of surgery needed to improve hearing.

Congenital defects of the ear can be catalogued into three clinical groups depending upon the severity of the anomalies.

Class I—Ears with the least abnormalities are listed in this group. The chief features are:

1. Usually normal auricle, or slightly smaller than normal.

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† Department of Otolaryngology, University of Illinois College of Medicine, Illinois Eye and Ear Infirmary.

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2. Partial, incomplete stenosis of the external auditory canal.

3. Pale eardrum, malleus handle is avascular and appears chalky.

4. Conductive hearing loss which may reach 60 db in the speech frequencies.

5. Mastoid process usually well-developed and pneumatized.

Class II—This group includes the majority of clinical cases of congenital ear defects and is characterized by:

1. Microtia.

2. Complete bony or membranous stenosis of the external auditory canal.

3. Absent drum.

4. Conductive hearing loss usually to 60 db for the speech frequencies.

5. Mastoid process pneumatized.

6. Ossicles normal or deformed.

7. Other abnormalities of a minor nature of the middle ear.

Class III—Ears with the most severe abnormalities are found in this group. Seen in this type of congenital disorders are:

1. Microtia.

2. Complete stenosis.

3. Absent drum.

4. Sclerotic, underdeveloped mastoid process.

5. Severe deformity or absence of the ossicles.

6. Gross deformities or absence of the middle ear.

In 34 patients of all types operated, severe anomalies of the facial nerve pathway have been observed only in this group.

We shall confine our discussion and presentation chiefly to the Class I, or least severe, type of deformities.

For over 20 years, one of us (R.H.) has noted in over 2500 cases of temporal bone surgery, that minor anomalies

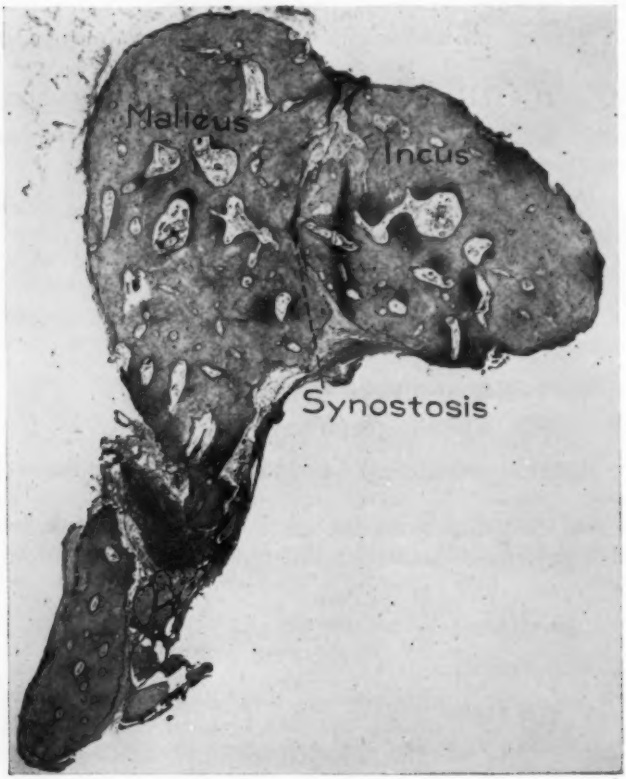


Fig. 1. Low power view of synostosis associated with cholesteatoma.

of the ossicular chain are often found in association with small attic cholesteatomas. These ossicle anomalies were noted most frequenty in children, as extension of the disease

in adults is often severe enough to erode the incudo-malleolar joint.

When care is used to remove the incus and head of the malleus together, and serial histologic sections of the ossicles

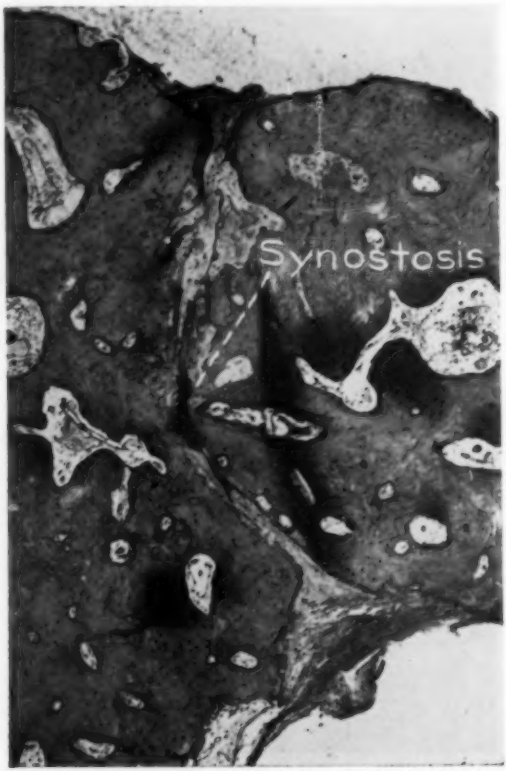


Fig. 2. High power view of Fig. 1, showing bony bridge between malleus and incus.

are made, synostosis and synchondrosis between the incus and malleus are often found (see Figs. 1 and 2). Juers[1] has reported one such instance.

This observation of an apparent developmental anomaly of the ossicles associated with cholesteatoma leads us to believe that in this type of case, both ossicle deformity and cholesteatoma have a congenital etiology. This opinion is forti-

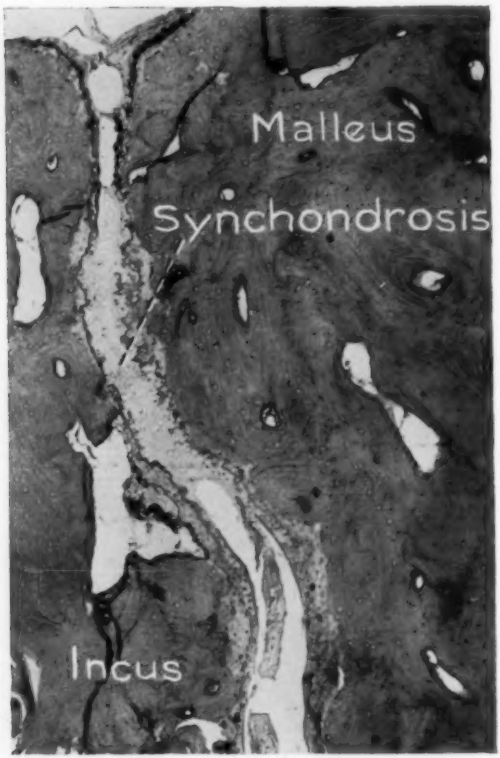


Fig. 3. Case 1. Synchondrosis, high-power view showing cartilaginous union between malleus and incus.

fied by the observation that superimposed otitis media usually results in attic perforation, whereas uncomplicated acute otitis causes central perforation.

The reported results of other otologists and our own experience has shown that surgery for removal of small cholesteatomas of the attic and antrum is highly successful in eradicating disease and preserving hearing. The cholesteatoma can be removed and further destruction of the conductive apparatus stopped and the hearing often improved over the preoperative level.

Because of these successes in congenital deformity with cholesteatoma and because of our interest in the surgery of major congenital ear deformities, our attention was drawn to those cases of unilateral complete atresia and a partial stenosis of the opposite canal and a bilateral conductive hearing loss. We felt that some of these patients might be deafened in the ear without atresia by the presence of an ossicular impedance. When Case 1, reported below, presented himself, we felt justified in attempting surgical exploration and removal of the impedance. We were aware that our results would probably not exceed the 30 db level of an interrupted ossicular chain.

Case 1: D. D., a nine-year-old white male, was first seen at the Illinois Eye and Ear Infirmary in October, 1953. History and examination showed a recurrent left mastoiditis. The hearing in the left ear was slightly impaired. An extensive simple attico-mastoidectomy was performed with good results. During the initial examination, it was noted that there was an average 60 db loss in the speech frequencies of the right or "uninvolved" ear.

Bone conduction studies revealed normal cochlear reserve bilaterally. The right external canal was partially stenosed and presented a constant problem with cerumen accumulation. The malleus handle appeared avascular and chalky white. The drum was dull. There was no fluid in the middle ear on the right. X-rays showed a normally pneumatized mastoid process.

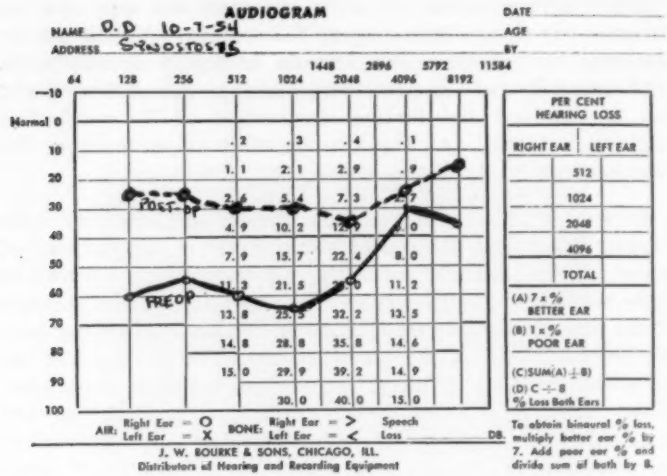
On Oct. 7, 1954, an exploratory surgical procedure was performed on the right ear to determine whether the hearing could be improved by interrupting the impeded ossicular chain and at the same time, enlarging the partial stenosis of the external canal.

An endaural fenestration type tympano-meatal flap was elevated, bony stenosis of the canal removed with the mastoid burr and the lateral attic wall removed. A complete mastoidectomy was performed.

When the ossicular chain was inspected with the microscope, the incus was found to be smaller than normal and completely fused to the head of the malleus.

The incudo-stapedial joint was carefully disarticulated and the neck of the malleus severed and the synostotic and synchondritic ossicles removed together (see Figs. 3 and 4).

Case 1.


AUDIOGRAM
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DATE
AGE
BY
64 128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
Normal 0
POST-OP
PRE-OP
AIR: Right Ear = O Left Ear = X
BONE: Right Ear = > Left Ear = <
Speech Loss DB.
J. W. BOURKE & SONS, CHICAGO, ILL.
Distributors of Hearing and Recording Equipment
PER CENT HEARING LOSS
RIGHT EAR
LEFT EAR
512
1024
2048
4096
TOTAL
(A) 7 x % BETTER EAR
(B) 1 x % POOR EAR
(C) SUM(A) ÷ 8
(D) C ÷ 8 % Loss Both Ears
To obtain binaural % loss, multiply better ear % by 7. Add poor ear % and divide sum of both by 8.

Case 2.


AUDIOGRAM
NAME E.T. 11-9-54
ADDRESS (SYNOSTOSIS - TREACHER-COLLINS)
DATE
AGE
BY
64 128 256 512 1024 1448 2048 2896 4096 5792 8192 11584
Normal 0
POST-OP
PRE-OP
AIR: Right Ear = O Left Ear = X
BONE: Right Ear = > Left Ear = <
Speech Loss DB.
J. W. BOURKE & SONS, CHICAGO, ILL.
Distributors of Hearing and Recording Equipment
PER CENT HEARING LOSS
RIGHT EAR
LEFT EAR
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TOTAL
(A) 7 x % BETTER EAR
(B) 1 x % POOR EAR
(C) SUM(A) ÷ 8
(D) C ÷ 8 % Loss Both Ears
To obtain binaural % loss, multiply better ear % by 7. Add poor ear % and divide sum of both by 8.

The tympano-meatal flap was then laid over the excavated mastoid as done in the fenestration operation.

The postoperative hearing gain was significant. Hearing for the speech frequencies averaged 30 db (see Fig. 5). Encouraged by this result, the next case was attempted.

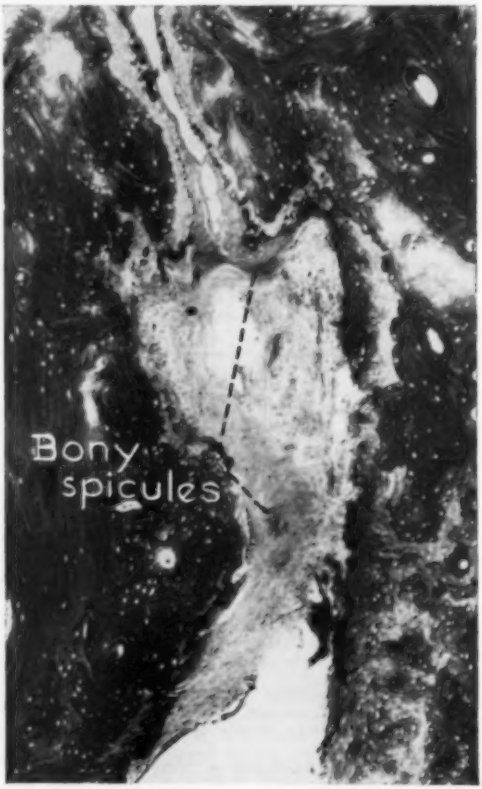


Fig. 4. Case 1. Synostosis, Mallary stain showing bony spicules between malleus and incus which appears as deeply stained tissue.

Case 2: E. T., a six-year-old white male, was referred to the Illinois Eye and Ear Infirmary because of poor speech and hearing. Patient was born with multiple congenital defects of the Treacher-Collins type and had had multiple corrective plastic procedures to the face and jaws and eyelids. In addition to the defects of the face, examination showed

partial stenosis of both external auditory canals. The ear drums were normal, except for the typical avascularity of the drums.

Audiometric studies showed an average loss in the speech range of 55 db in the right and 50 db in the left. Bone conduction was normal bilaterally.

Mastoid X-rays showed limited development bilaterally.

A hearing aid was advised, but it was difficult to fit an air conduction aid to the stenotic canals.

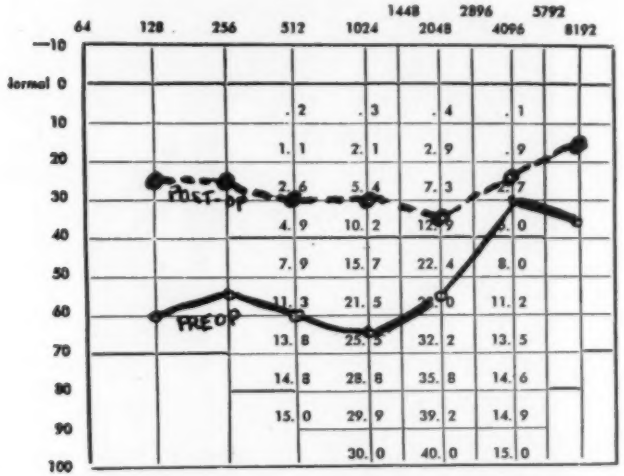


Fig. 5. Audiogram, Case 1, Pre- and post-operative hearing. Dotted line indicates post-op. thresholds.

A modified radical mastoidectomy was performed Nov. 9, 1954, to correct the stenosis of the canal and to explore the middle ear since a Class I type of ossicular deformity was suspected.

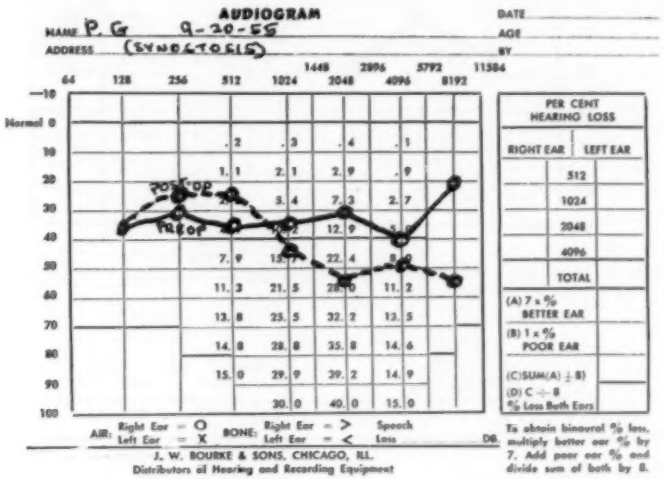
An endaural incision was made and a tympano-meatal flap cut. The bony partial stenosis was removed, the lateral attic wall removed, and a complete mastoidectomy performed. A synostosis of the malleus to the incus was found and the synostotic ossic'es removed. The tympano-meatal flap was laid down and packed as usual.

On Jan. 6, 1956, an audiogram showed an average hearing of 30 db for the speech frequencies in the right ear (see Fig. 6).

The next case occurred in the right ear of a patient in whom we had achieved a successful hearing improvement in the left ear which was impaired with a complete atresia of the Class II type.

Case 3: P. G., a five-year-old female, who had been operated on for the above-discussed Class II left congenital atresia, was found to have a right hearing loss averaging 35 db. This patient had repeated episodes of acute otitis media in this ear which occasioned an additional fluctuating hearing loss. The associated narrowing of the external canal and the accumulation of cerumen made it difficult to treat the acute otitis media. During an apparent remission of middle ear disease, corrective surgery was undertaken on Sept. 20, 1955. At surgery, a tympano-meatal

Case 3.



flap was cut and the canal was widened. The mastoid process was entered and the entire cellular system was filled with granulations, but no bone necrosis was present. The lateral attic was removed and the mastoidectomy was enlarged to a complete atticotomy exposing the ossicular chain. The incus and malleus were small, deformed, and fused. The incudo-stapedial joint was separated, and the neck of the malleus clipped, and the incus and head of the malleus were thus removed. The flap was then laid in place as in a fenestration operation and the wound packed. Her resultant hearing averages 44 db. (See Fig. 7). This result is confused by the coexisting mastoid disease; however, we do feel that defects which border the practical level of hearing do not warrant interrupting the impeded ossicular chain.

Case 4: S. M., age eight-and-one-half years old, has been observed since May 11, 1953, and presents a very mild Treacher-Collins syndrome on the left only. This side has aplasia of the temporo-mandibular joint, polyotia in the area of the tragus, and a conductive hearing loss on this side averaging 40 db. The external canal is mildly stenotic, the drum is dull,

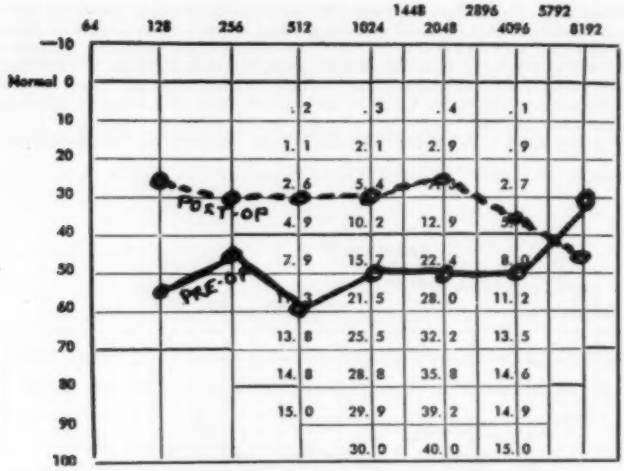


Fig. 6. Audiogram, Case 2. Pre- and post-operative hearing. Dotted line indicates post-op. thresholds.

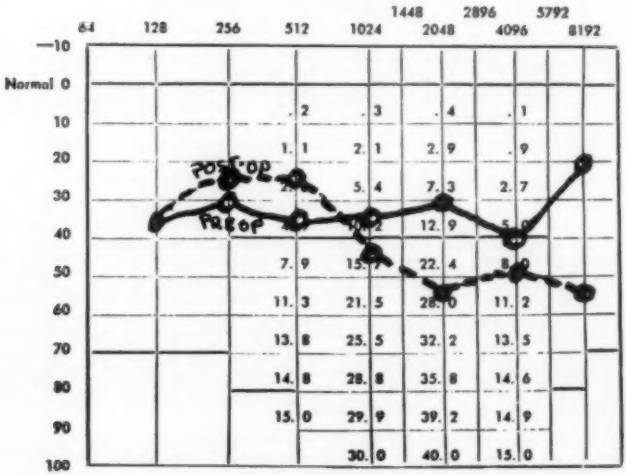


Fig. 7. Audiogram, Case 3. Pre- and post-operative hearing. Dotted line indicates post-op. thresholds.

and the hammer handle is avascular. Because of the associated congenital deformities, we suspect the hearing loss in this ear to be the result of impedance of the ossicular chain, probably due to synostosis; however, because of the completely normal hearing on the right ear and small expected gain from surgery, we have not operated this patient.

DISCUSSION.

Congenital synchondrosis and synostosis of the ossicular chain are regularly seen associated with stenosis of the external auditory canal. H. Marks,[2] in a survey of the litera-

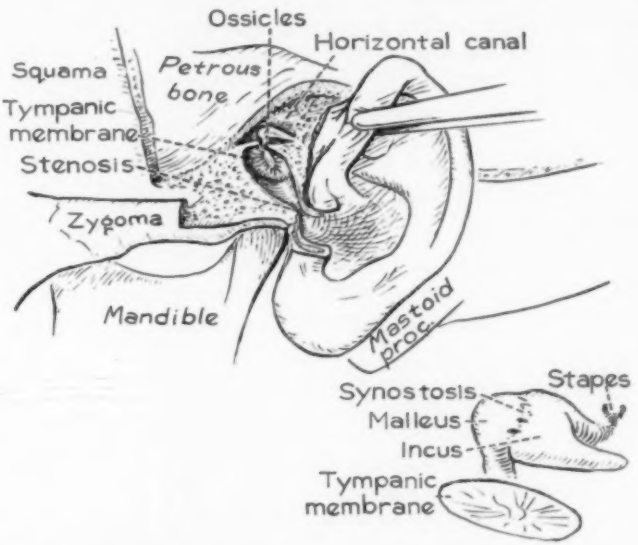


Fig. 8. Diagrammatic sketch showing stenosed external canal associated with synostosis of ossicles.

ture, found that in forty-eight reported cases of congenital stenosis of the external canal, only two ears had normal adult ossicles. F. Altman has reported ossicle deformities in almost all of the severely congenitally deformed ears that he has studied.

In our own thirty-four cases, only three patients had adult-

appearing and mobile-enough ossicles to merit non-removal at the time of surgery.

We wish to point out that the ossicular deformities may be present alone or in association with mild stenosis of the external canal and cause handicapping hearing losses that may be improved significantly (see Fig. 8).

Such isolated anomalies are rare, but careful search for the diagnostic criteria presented should reveal more cases of this type. H. House[3] recently reported two cases of fusion of the incus to the tympanic ring as the only apparent defect of the complex conductive hearing mechanism, and excellent hearing improvement resulted after freeing the impedance of the ossicular chain.

SUMMARY.

1. Minor congenital ossicle defects of synostosis and synchondrosis of incus and malleus may cause severe conductive hearing loss.

2. A classification of congenital aural defects is presented, and attention is drawn to the common association of cholesteatoma and ossicular defects.

3. Three proven cases of synostosis of the malleus and incus as the sole cause of conductive hearing loss and surgical correction are presented. A fourth probable case is presented.

4. Minor congenital ossicular anomalies may cause severe conductive hearing losses; these ears may be microtic; the canal may be mildly stenotic; the drum appears dull, and the hammer handle of the malleus appears chalk-like and avascular.

5. This type of anomaly is probably more common than is realized. Careful search should find more cases of this type.

6. Altman[4] in his excellent report of a case of congenital atresia of the ear observed an osseous connection between the stapes and the facial canal. He stated that the chances for improvement of hearing are doubtful due to the frequency of bony bridges between the stapes and the medial tympanic wall

and other minute deformities which might escape detection. While the results do not always meet our expectations, meticulous technique, the use of the operating microscope, and newer Cortisone-like drugs which decrease inflammation and scar tissue, encourage us to continue in our efforts to improve surgically the congenital conductive hearing loss patient.

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DR. CHARLES BRAMMAN MEDING AWARD.

On May 21, 1956, at The Harlem Eye and Ear Hospital, Dr. Eugen Grabscheid received the Dr. Charles Bramman Meding Award. This award, granted annually in memory of Dr. Meding, was bestowed upon Dr. Grabscheid in recognition of his outstanding clinical service at The Harlem Eye and Ear Hospital and for his presentation of programs of study in the interest of the advancement of the art of Otolaryngology.

UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Ill.

SURGICAL TECHNIQUE OF REMOVAL OF INFRATEMPORAL MENINGIOMA.

JOHN J. CONLEY, M.D.,*

GEORGE T. PACK, M.D.,*

and

SALVADOR S. TRINIDAD, M.D.,†

New York, N. Y.

Meningiomas causing exophthalmos from their intra-orbital placement may originate from several sources, *viz.*, *a.* anterior cerebral meninges, *b.* meningiomas of olfactory groove passing through the foraminal narrows (Cushing), *c.* meningeal tumors lying within and expanding the vaginal sheath, *d.* sphenoidal ridge tumors growing into the orbit and *e.* meningiomas producing tumor-hyperostosis from invasion of the orbital bones. Those meningiomas not arising from the sheath of Schwalbe and not involving the optic nerve proper with atrophy and blindness usually are located in the lateral orbital compartment. Cushing[1] directed attention to the tendency of meningiomas to expand in the line of least resistance and "to crowd their way into all neighboring anatomical crannies and pockets."

In the patient who is the subject of this case report, the meningioma presented itself chiefly in the infratemporal fossa, having extended there from the left lateral orbit. The lateral wall of the bony orbit was invaded by the tumor. The optic nerve and sheath of Schwalbe were grossly free of evidence of tumor. Psammoma bodies within the tumor aided in the identification of its histogenesis. The histological findings in these extracranial meningiomas are consistent with the hypothesis that all of them may be intracranial in origin even though the bulk of the tumor is without the skull. In this

* From the Pack Medical Group and the Surgical Service, Doctors Hospital, New York City.

† From the Department of Pathology, Doctors Hospital, New York City.

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patient, there are no symptoms, no physical findings and no radiographic evidence of intracranial involvement, but the disease will be suspected until a long period of postoperative observation furnishes convincing proof otherwise.

CLINICAL HISTORY.

H. M., a 45-year-old white male, related that he had been aware of a gradual swelling in the region of the left temple and increasing prominence of the left eye for two months prior to his first visit to this clinic on April 7, 1955. There were no symptoms related to this mass.

Clinical examination revealed a deeply fixed mass completely filling the left infratemporal fossa and protruding in the region of the temple just above the left zygoma. The left eyeball was proptosed 2 mm., but vision, extra-ocular movements, and fundoscopic examination were normal. There was no trismus, no cranial nerve involvement, and X-rays of the skull with particular attention to the infratemporal fossa and orbital regions were unrevealing.

Aspiration biopsy was interpreted as a probable malignant tumor in the infratemporal fossa and retro-orbital region. Lymphoma, primary somatic tumor, neurogenic tumor and tumor of the lacrimal gland were also considered.

Because of the indefinite histogenetic diagnosis afforded by the aspiration biopsy and the clinical features simulating the Mikulicz lymphoma syndrome, a therapeutic test by X-ray therapy was started. A tumor dose of 4200 r. was given during three weeks of irradiation using the following factors: 250 KV, 50 cm. T-S distance, 4 cm. cone, 2 ports, filter of 1.5 mm. Cu. The regression was not typical of lymphoma within the customary period of post-irradiation observation; therefore, surgical exploration was advised.

TECHNIQUE.

The skin incision began in the medial part of the left eyebrow, curving inferiorly and laterally to a position just above the zygoma and then posteriorly to the superior attachment of the ear. Here it joined a vertical excision extending from the region of the temple down to the level of the tragus. The skin flaps were elevated widely down to the superficial temporal fascia, thus affording an excellent view of the lateral bony orbit, zygoma and temporal muscle. The zygomatic bone was completely exposed along with the zygomatic extension of the temporal bone by cutting the origins of the masseter, quadratus labii superior, and zygomaticus muscles. The temporal segment of the zygomatic arch was green-stick fractured posteriorly. The remainder of the overhanging zygomatic bone was removed with appropriate bone-cutting rongeurs. A firm fixed mass could be felt filling the entire

infratemporal fossa and causing a bulge in the temporal muscle at this level. The temporal muscle was then transected 2 cm. above the coranoid process of the mandible, and the

Fig. 1-A. Preoperative view showing protuberance in left infratemporal area and post-irradiation pigmentation.

tumor came into direct view. It contained an indefinite capsule and extended in the direction of the infra-orbital fissure and orbital cavity. The zygomatic portion of the lateral bony orbit

was then totally removed, thus uncapping the infra-orbital fissure and establishing a common exposure of the orbit and infratemporal fossa. The orbital portion of the tumor was

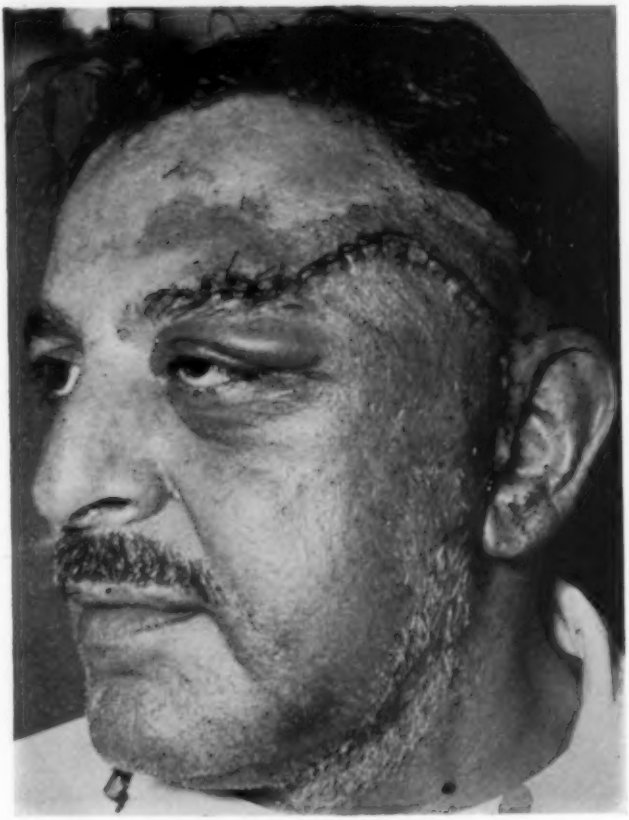

Fig. 1-B. One week postoperative.

attached to the infra-orbital fissure. From the primary attachment in the region of the infra-orbital fissure, the tumor extended medially and posteriorly over the great wing of the sphenoid. A small section of bone at this site was destroyed

by tumor. Once the exposure was complete, the tumor came out with surprisingly little difficulty. The maxillary nerve was resected, and the internal maxillary artery was ligated.

Fig. 1-C. Three months postoperative.

A portion of the pterygoid wing of the sphenoid was removed, and the dura appeared normal. The wound was then closed in layers by first approximating the cut segments of the temporal muscle and the subcutaneous tissue with fine silk. The

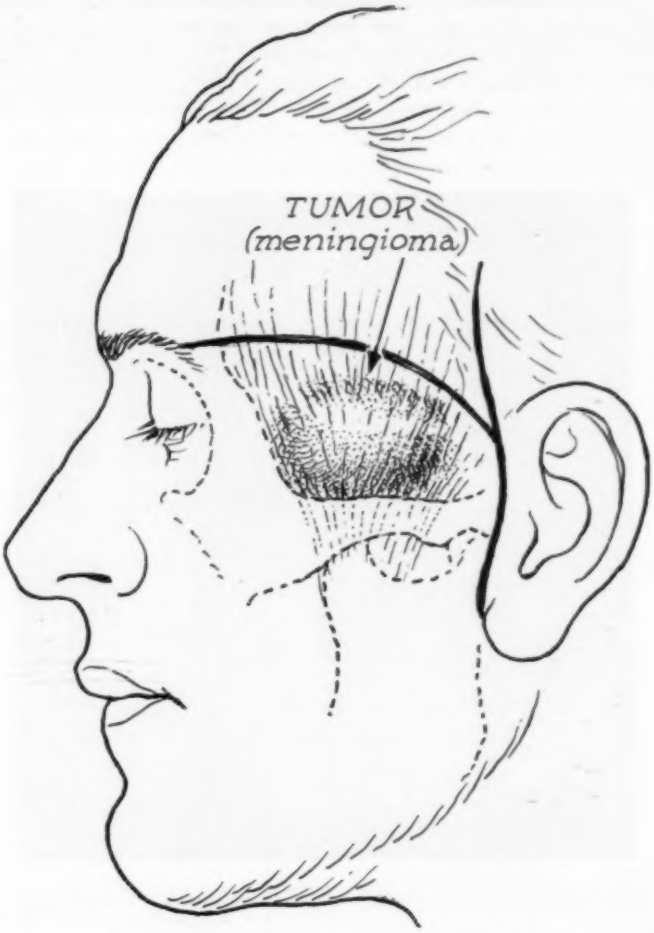

Fig. 2. Meningioma situated in the infratemporal space.

residuum of the zygomatic portion of the temporal bone was placed into position and the skin flaps closed with silk, with a smaller rubber drain extending into the temporal fossa.

The postoperative course was uncomplicated. The patient complained of a left-sided headache for two days postoperatively, which required mild analgesia. The periorbital edema subsided gradually within two weeks. The extra-ocular movements of the eyeball were normal. There was no diplopia and

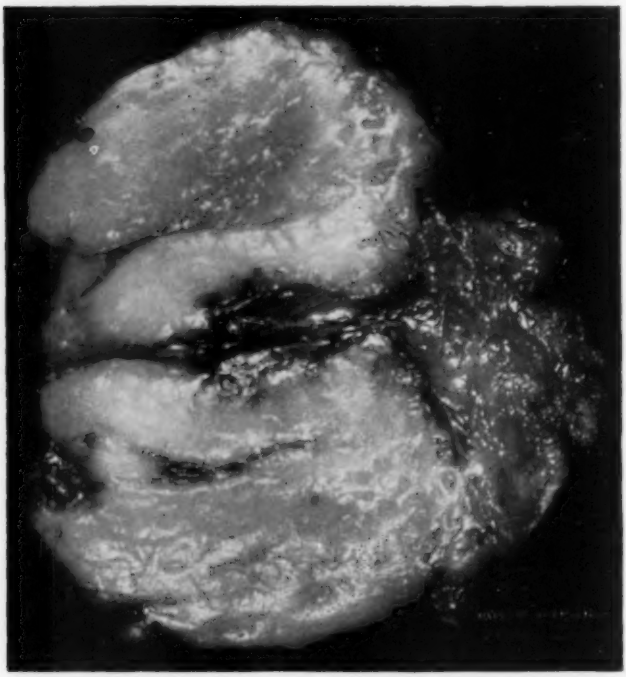

Fig. 3. The tumor of the temporal fossa is homogeneous in appearance.

no proptosis. There was an area of anesthesia in the region of the infra-orbital nerve. Mastication was slightly weakened on the left side but was adequate. The drain was removed on the third postoperative day, and the wound healed per primum (see Figs. 1 and 2).

MICROSCOPY.

The tumor measured 4 x 3 x 2 cm. and was lobulated, pink-gray, and moderately firm. The cut surface was dense, homogeneous, and tan-gray (see Fig. 3).

The tumor was characterized by a whorled pattern of fibrillary cells of homogeneous appearance. The whorls were

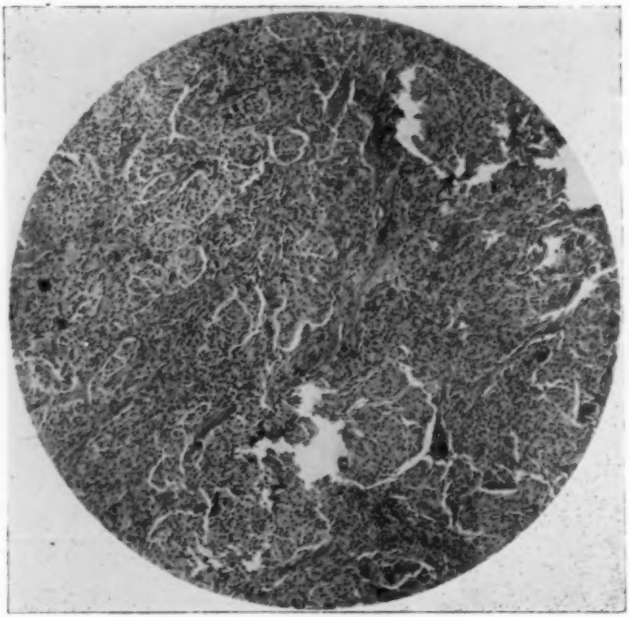

Fig. 4. The fibrillary masses are separated by bands of fibrous tissue producing a lobulated pattern. The masses have a homogeneous appearance and the nuclei are large and oval. X 60.

separated by relatively narrow bands of hyaline connective tissue. The nuclei were large and oval. Occasionally, individual nuclei were large and deeply stained. Mitoses were absent (see Fig. 4). At the periphery, the tumor tissue penetrated into the adjacent muscle. At this site, the nuclear

pattern displayed more anisocytosis than the main mass. The vessels were dilated and small, edematous foci were numerous.

The marrow spaces of the temporal bone were filled with tumor tissue of similar nature. In addition, there were several Psammoma bodies (see Fig. 5). The specimen was diagnosed as invasive meningioma.

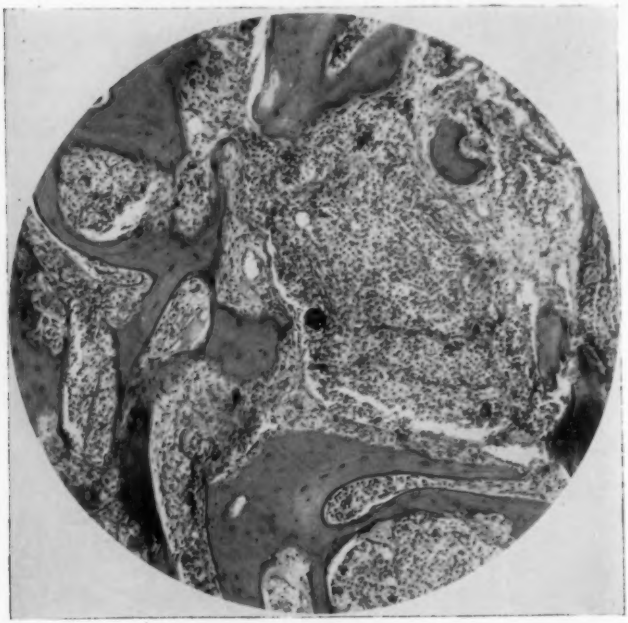

Fig. 5. The marrow spaces are filled by tumor tissue of whorled pattern. A psammoma body is seen in one area. X 90.

SUMMARY.

An unusual case of extracranial meningioma involving the infratemporal and orbital regions was removed by a direct approach to the infratemporal space. The direct approach gave better exposure of this region than the usual scalp flap.

The eye was not affected and the postoperative course was uneventful.

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139 East 36th Street.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M.D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

WASHINGTON UNIVERSITY, SAINT LOUIS, DEPARTMENT OF OTOLARYNGOLOGY.

Offers a Basic Science Course in Otolaryngology to start Monday, September 17, 1956. Complete information about the course may be obtained by writing to Theo. E. Walsh, M.D., Head of the Department of Otolaryngology, 640 S. Kingshighway, St. Louis 10, Missouri.

RELATIONSHIP BETWEEN ETIOLOGY OF HEARING LOSS AND RESULTANT AUDIOMETRIC PATTERN.*

W. W. WILKERSON, JR., M.D.,
and
JONATHAN I. H. DOYLE, M.A.,

Nashville, Tenn.

THE PLAN OF STUDY.

The Origin of the Cases.

The original data for this study was derived from the pure tone audiograms of 175 hearing impaired children in the age range from six to 14 years, all of whom had been referred to the Hearing and Speech Center† for Audiological Evaluation during the 21 month period ending April 1, 1953. Because of meager information, either in the medical history or the informant's report in the initial interview, 24 audiograms were discarded, leaving a total of 151 for detailed study and analysis.

Each of these 151 children was referred to the diagnostic clinic by an ear, nose, and throat specialist, with an accompanying otological report.

The audiological assessment at the Center consisted of detailed hearing testing, comprising both pure tone and speech audiometry, complete case history, and testing in allied areas, including intelligence screening, when indicated. Each pure tone audiogram was done by a trained audiologist in a sound proof testing chamber. For the pure tone testing, four different commercial audiometers were used, but each was methodically maintained in calibration with reference to average normal threshold.

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* Read at the Meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., Houston, Tex., Jan. 27-28, 1956.

† Research work of the Staff of the Bill Wilkerson Hearing and Speech Center.

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THE METHOD OF CLASSIFYING AUDIOGRAMS.

In any study involving the grouping together of a large number of audiograms, it is essential that they be classified in a definite and simple manner. Several methods have been contrived in the past 20 years. One of the earlier was devised by Guild[1] in 1932. His emphasis was on the main characteristics of the plotted line; that is, the more general form, direction, and position.

In the interest of the United States Public Health Service, Ciocco[2] in the early thirties, began a survey of the hearing of almost 1400 school children. Ciocco's method of classification included eight basic categories.

A more recent method was devised by Carhart[3] to meet the needs of the acoustic clinic at the Deshon Army Hospital during World War II. His aim was to achieve a coding procedure which would be sufficiently inclusive in scope and universal in application.

The categories of audiogram patterns devised for the present study were not based on any one previous method, but salient features from each of the methods suggested by Ciocco and Carhart were adapted and integrated into the final plan. The outcome was the following eight classifications, which seemed to provide for the various deviations encountered in the audiograms, and at the same time afforded the simplicity of description shown by Ciocco and the definitive scope of Carhart:

Group 1. Good hearing for all tones. All tones heard at an intensity equal to or less than 20 db at six of the seven frequencies tested.

Group 2. Near normal hearing at the two lower octaves of the speech frequency range with precipitous drop in acuity at 2,000 cps. or above.

Group 3. Gradual downward slope with progressively greater loss for higher frequencies at a slope of five to ten db per octave across the audiogram.

Group 4. Marked high tones loss, characterized by at least one 20-db slope per octave; within the 250 to 2,000 cps. range,

or by no hearing within the audiometer limits for at least two of the frequencies above 1,000 cps.

Group 5. Moderate loss for all tones; thresholds between 20 and 65 db for at least six of the seven frequencies of the audiogram.

Group 6. Marked loss for all tones; thresholds between 65 and 100 db for at least four of the five middle frequencies (250 to 4,000 cps.).

Group 7. Rising audiometric contour with general pattern of loss for frequencies below 1,000 cps. ten to 30 db greater than for tones above 1,000 cps.

Group 8. Trough-shaped curve with greatest depression occurring in speech frequency range.

THE METHOD OF CLASSIFYING ETIOLOGIES.

Since the initial categorization was based on the audiometric curve, the various etiologies determined for each subject's hearing loss were at first merely listed audiometric pattern groups. In order to tabulate data for inspection and analysis, it soon became evident that the causative factors, like the audiogram curve, needed to be classified into the smallest number of categories feasible.

This problem presented some difficulty because of the many known causes of hearing impairment. The chief source of information for determining the etiology was the otological examination and the case history. When no cause whatever could be found, the etiology was noted as "cause undetermined."

Otitis media and hypertrophied tonsils and adenoids were combined to form one group, since these conditions are somewhat alike in their initial effects upon the hearing mechanism. Hypertrophied tonsils and adenoids, and otitis media cause a fairly uniform reduction in acuity for all tones.

Such diseases as influenza, meningitis, pneumonia, mumps, measles, virus, and whooping cough may involve the VIIIth nerve or its terminal branches. In view of this fact, they lend themselves to a single classification, which Fowler[4] has spoken of as inflammatory neuritis.

Loss of hearing due to heredity, birth injury, rubella, prematurity, drugs, and head injury is known to cause perceptive hearing loss. Still it was felt that for purposes of this study they needed separate classification, because each condition may have a selective action on certain segments of the auditory neural mechanism. Unfortunately, the number comprising these groups was in several instances quite small.

The classification of psychological etiology was used when marked discrepancy between the speech and pure tone results was found to exist. These cases also evidenced functional hearing within normal limits, although the pure tone audiometric evaluation indicated a decrement in acuity. The evidence of normal hearing was in each instance established conclusively by the use of speech audiometry.

TABLE I.

INCIDENCE OF ETIOLOGIES OF HEARING LOSS ACCORDING TO AUDIOMETRIC PATTERN.

Etiology	Audiometric Pattern Groups I	II	III	IV	V	VI	VII	VIII	Total
Otitis Media; Infected T. & A.	21	2	6	3	16	3	15	2	68
Inflammatory Neuritis	3	2	5	6	4	3	2	5	30
Heredity	0	0	2	3	5	2	1	1	14
Birth Injury	0	0	2	8	0	0	0	0	10
Psychological	0	0	1	0	4	1	3	0	9
Cause Undetermined	1	3	0	2	0	1	0	0	7
Rubella	0	0	1	0	2	1	0	2	6
Prematurity	0	1	0	2	1	0	0	1	5
Drugs	0	0	0	0	0	1	0	0	1
Head Injury	1	0	0	0	0	0	0	0	1
Total	26	8	17	24	32	12	21	11	151

The incidence of the various causes of hearing loss in the different audiometric pattern groups in shown in Table I. The information presented in this Table forms the basis for the detailed analysis of audiogram patterns in this study.

INFORMATION YIELDED BY AUDIOGRAM PATTERN GROUPING.

Group I. Good Hearing for All Tones. Fig. 1 represents the mean air and bone conduction curves for the 26 children in the Group I category, which includes good hearing for all tones with hearing as good as 20 db for six of the seven frequencies tested in each individual case.

Twenty-one, or 80.8 per cent of the 26 cases were thought to have this slight hearing decrement as a result of either otitis media or infected tonsils and adenoids. In a number of instances the children in Group I may have received otological care which improved their hearing status prior to the audiological testing at the clinic. This situation occurred with seven of the 26 cases in the group.

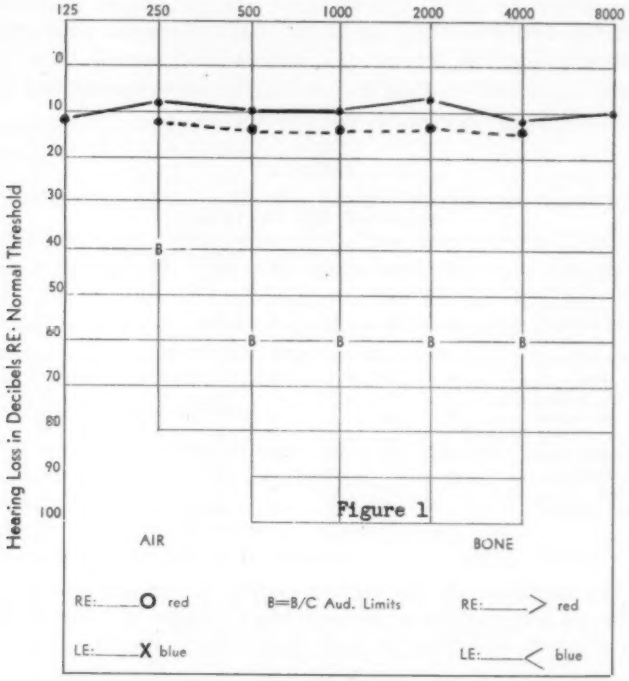

Figure 1

The fact that as many as four of each five audiograms in Group I were associated with an etiology of otitis media, or hypertrophied or diseased tonsils and adenoids, is considered significant. The mildly depressed air conduction curve throughout the frequency range is the type of hearing pattern which

beginning middle ear difficulty may often be expected to present.

Group II. Near Normal Hearing in Low Frequencies with Sharp Drop-off in High Frequencies. Only eight of the total number of audiograms analyzed fell in the Group II configuration, which indicates near normal hearing at the 500 and

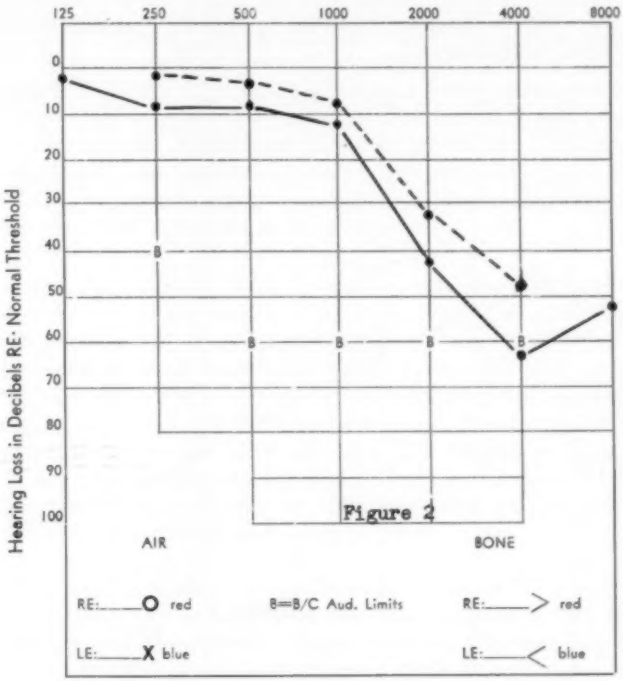

Figure 2

1,000 cycle tones, but with a precipitous drop in acuity beginning at 2,000 cps. or above. Persons with this type of hearing loss, represented audiometrically by the mean curve in Fig. 2, were unable to hear many of the high frequency consonant sounds, such as (s), (sh), and (th) in everyday conversational speech.

It is noted that the eight included in this classification were rather randomly distributed as to the causative factor. For three, or 34.5 per cent, no cause could be determined.

The closely parallel relation between the mean bone and air curves, as shown in Fig. 2, would seem to indicate that this type of pattern is the result of nerve damage to particular segments of the basilar membrane. The random distribution of etiologies and the small number in this group make any further statement as to the cause of this type of hearing impairment unwarranted.

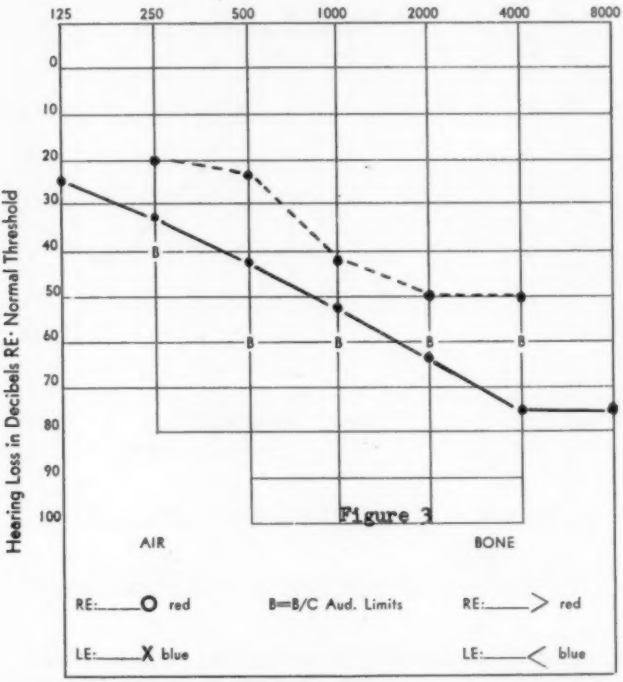

Figure 3

Group III. Gradual High Tone Loss. The configuration revealed by Group III, presented in Fig. 3, denotes a serious hearing problem, with the gradual downward slope increasing

about ten db per octave from left to right. The loss in acuity throughout the speech frequency range denotes an inability to hear most of the consonant sounds in conversational speech. Seventeen children, or 11.2 per cent of all the cases, were in this category. Six of 17, or 34.3 per cent of the group, were associated with otitis media, or infected or enlarged tonsils and adenoids. These findings supported the fact that chronic middle ear conditions often may eventually result in a mixed-type loss which is irreversible in large part because of the extent of perceptive impairment. The slight conductive component of the mean audiogram is contributed chiefly by the six mentioned above. Five, or 29.4 per cent, were associated with inflammatory neuritis from mumps, measles, whooping cough, and meningitis. Inspection of their individual audiograms showed that these cases were all of the perceptive type. Two birth injury, two heredity, one rubella, and one psychological were the other etiologies in this pattern.

Group IV. Marked High Tone Loss. The mean audiometric curve for Group IV, seen in Fig. 4, presented a marked downward slope within the speech frequency range, with a severe depression in the higher frequencies. Each audiogram chosen with this pattern had at least one per-octave drop as great as 20 db in the 250 to 2,000 cps. octave range. The mean air conduction threshold for 1,000 cps. was 25.4 db greater than for 500 cps. This octave band appeared to be the one which is most often characterized by this essential feature of the marked high tone loss audiogram.

This type of pattern was evidenced by 24, or 15.9 per cent of the total 151 audiograms, and was, therefore, the third largest group numerically. Eight children, representing one-third of the group, gave a history of birth injury, suggesting that this etiology has a selective effect upon the acuity for high frequency sounds.

(Twelve additional cases of birth injury were studied after the completion of this paper. Ten of these cases had a hearing loss of less than 20 db at 125, 250 and 500 cps., with a sharp drop for the higher tones.) The auditory acuity of the other two cases followed the pattern shown in Fig. 4. Six children,

or one-fourth of the number in the group, had hearing loss attributed to inflammatory neuritis associated with meningitis and childhood exanthemata. Two or three each were distributed randomly among all the etiologies except rubella, psychogenic, drugs, and head injury.

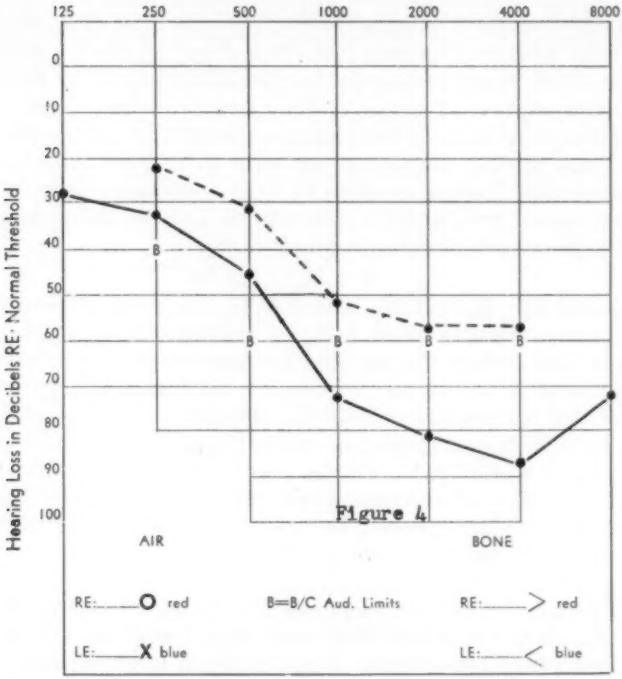

Figure 4

Group V. Moderate Loss for All Tones. The audiograms making up Group V represented a moderate loss for all tones. Thirty-two, or 21.2 per cent of the total number of audiograms studied constituted this category, which comprised the largest numerical group of the eight major audiogram pattern classifications. Fig. 5 revealed a fairly uniform loss throughout the

frequency range, with all mean thresholds except that for 125 cps., lying between 40 and 50 db. It was significant to note that 16, or 50 per cent of the subjects, in this group, were referred to the Hearing Center with otitis media or infected or enlarged tonsils and adenoids. This fact accounted for the substantial conductive component shown by the mean audio-

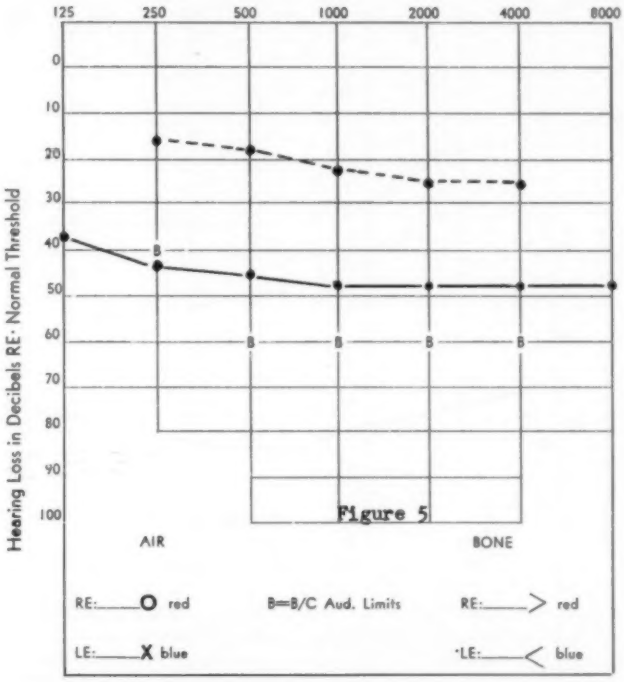

Figure 5

gram air-bone gap. Seven, or 21.9 per cent, of these cases were diagnosed as either rubella or heredity. The incidence of these conditions is known to be reflected audiometrically by perceptive type involvement, which would be the factor that would depress the bone conduction curve in Fig. 5.

Group VI. Marked Loss for All Tones. Group VI was rep-

resented by 12, or 7.9 per cent of the total number of audiograms studied. This pattern was characterized by marked loss for all tones. Children in the group are classified as profoundly deaf. Because bone conduction testing has a 60 db intensity limit, the bone conduction curve was falsely weighted with those subjects having better hearing than 60 db for bone conduction tones. In order to include these with poorer hear-

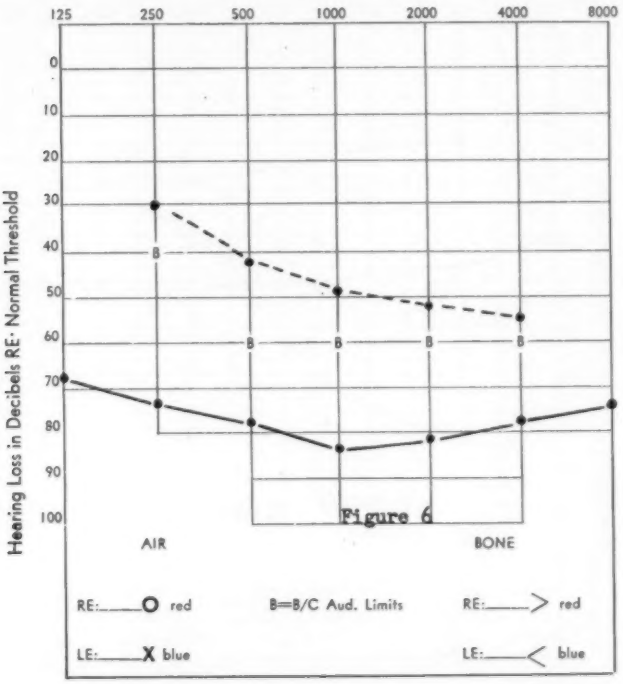

Figure 6

ing, thresholds were recorded at 60 db for all cases for whom the hearing level was actually poorer, but where true thresholds could not be obtained because of the intensity limits of the audiometer. This limitation accounts for the fact that all the mean bone conduction curves for the study show mean threshold values which indicate somewhat more conductive component than might be expected.

The 12 cases are rather randomly distributed throughout all the etiologies, except for prematurity, birth injury, and head injury. Three cases each, making one-half the total, were associated with the otitis media and the inflammatory neuritis causes.

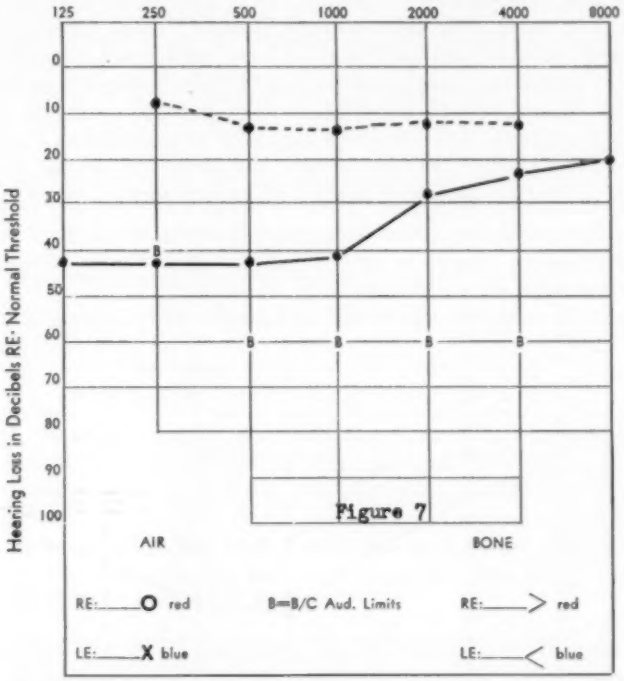

Figure 7

Group VII. Rising Audiometric Contour. In Group VII were found 21, or 13.9 per cent of the audiograms from the total number. This pattern was characterized by the rising audiometric contour, exactly opposite to the gradually downward slope denoting Group III. This type of profile rose gradually from 1,000 to 8,000 cps., with usually more uniform loss of hearing for the frequencies below 1,000 cps. (see Fig. 7). It was considered significant that 15, or 71.4 per cent of the audiograms displaying this pattern were associated with an

etiology of otitis media and/or infected or enlarged tonsils and adenoids. The presence of the nearly maximum air-bone gap shown by the mean audiogram, indicated that this pattern may be associated with the purely conductive type lesion. This is in agreement with what is known about the effects of the etiology which was so prominent in this group. The incidence of this pattern was noted once in association with heredity, and twice with inflammatory neuritis. These three audiograms may account for the mild bone conduction depression, since they were generally conceded to cause a nerve-type deafness. Three, or 14.3 per cent, were of psychological etiology. The child with functional loss may well be expected to display relatively greater inability to hear low frequency sounds, since they comprise the major portion of environmental sound.

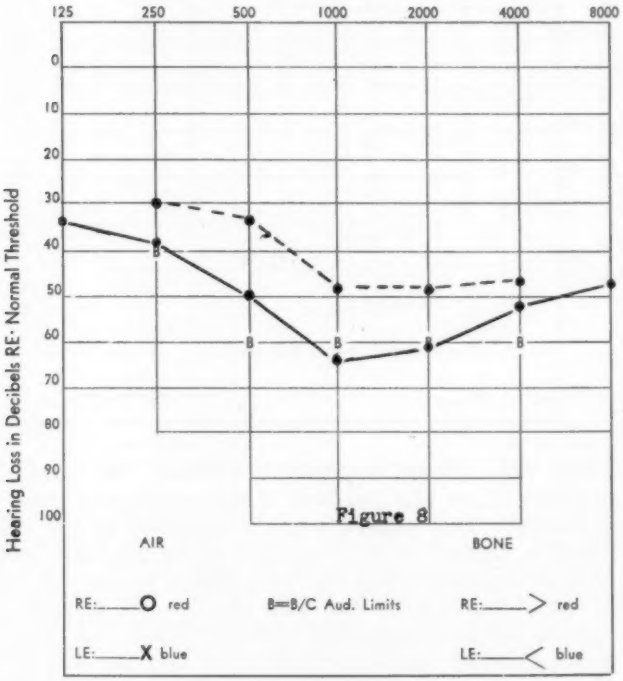

Figure 8

Group VIII. Trough-Shaped Curve. Eleven, or 7.2 per cent, of the total number of audiograms studied presented a trough-shaped curve, with the greatest depression occurring within the speech frequency range. Examination (see Fig. 8) showed that the higher frequencies above 1,000 cps. were affected more than those below 1,000 cps. This perceptive symptom was reasonable, in view of the fact that the largest percentages occurred in the rubella and inflammatory neuritis categories, being 18.2 and 45.5 per cent, respectively, since these two etiologies characteristically presented a nerve type loss. In six of the audiograms the point of greatest severity of loss was found to be at 1,000 cps., indicating this frequency may be the focus for the greatest damage in certain pathologies of hearing. The upper limit of the trough was at 20 db, while the point of greatest depression in any audiogram within the group occurred at 80 db. In nine of the 11 audiograms, the entire trough depression was centered in the speech frequency range, with thresholds occurring between 45 and 75 db.

INFORMATION YIELDED BY ETIOLOGIC GROUPS.

As stated previously, classification of data for the present study included a consideration of two factors, audiometric contour and etiology of hearing loss; therefore, it was necessary in a thorough analysis of the data to consider not only how the different etiologies were included in each of the eight patterns, but also how the different patterns were distributed among the ten etiological groups.

Sixty-eight, or 45 per cent of the 151 cases were diagnosed as otitis media or enlarged and diseased tonsils and adenoids. It was felt that this group was large enough to utilize the chi-square technique to determine the significance of the observed frequency distribution among the eight audiometric patterns. The chi-square value obtained was 41.312 which meets the one per cent level of confidence, indicating that the observed distribution of cases in this etiological group could not have occurred by chance factors alone, if there had not been some relationship between etiology and resultant audiometric contour.

It would appear that middle ear impairments caused by otitis

media and enlarged tonsils and adenoids may be significantly associated with both the "moderate loss for all tones" and the "slightly rising audiometric contour" audiograms.

The next largest etiological category was that of inflammatory neuritis, consisting of 30, or 19.9 per cent of the 151 cases distributed among the eight audiometric pattern groups. Sixteen, or 53.4 per cent of these 30 audiograms were characterized by gradual and marked high tone loss and trough-shaped curves. Each of these patterns represents primarily perceptive type loss.

The etiological classification of heredity ranked next numerically, containing 14, or 9.3 per cent of the total number of cases included. These audiograms revealed either a moderate loss at all frequencies, or marked high tone loss.

In the birth injury group were found ten, or 6.6 per cent, of all the cases. The fact that eight, or 80 per cent of these ten, revealed a marked high tone loss, appeared significant. Fisher's Exact Significance Test was used to compare the birth injury etiology distribution with all other etiology distributions. This test yielded a probability of .00001, indicating that the observed distribution of birth injury etiology, according to pattern, could not have occurred as a result of chance factors alone; therefore, the marked high tone loss audiogram may be significantly related to hearing loss resulting from birth injury.

Nine, or 6 per cent, of the cases were found to have functional hearing loss, and were in the "psychological" etiologic group. Audiograms of these nine were rather randomly distributed among all patterns of hearing loss.

Seven, or 4.6 per cent, were included in the category "cause undetermined".

Rubella occurred in six, or 4 per cent of the cases analyzed. Although the numerical representation for this etiologic group does not lend itself to statistical analysis, it is interesting to note that two-thirds of the rubella cases show moderate loss for all tones or trough-shaped curve. This confirms the findings of Bordley and Hardy.[5]

The incidence of prematurity is small, with five, or 3.3 per cent of the total number of cases in this etiologic group. Two of these five had audiograms characterized by marked high tone loss, and thus in contour resemble the type of curve noted to be significantly related to case of birth injury.

Two of the ten etiologic classifications were represented by only one case each. These two were "drugs" and "head injury". The small incidence prevents any elaboration on the type of audiogram pattern each showed.

In summarizing the interpretation of the data, it can be pointed out that the present study of relationships between audiometric configurations and etiologies of hearing loss has lent support to the concept that causes of hearing impairment can be more definitively related to audiogram patterns. A number of interesting relationships have been noted. Although only a few of these were treated for statistical significance, it is believed that a larger sampling of the various etiologic groups would yield results that could be substantiated.

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FENESTRATION OPERATION IN THE POOR CANDIDATES.*

44 Cases Selected From 637 Operations.

J. BROWN FARRIOR, M.D.,

Tampa, Fla.

INTRODUCTION.

The primary purpose of this paper is not to advocate fenestration surgery in the poor candidate, but to find out what has been done with the fenestration operations in these cases. I have gone through the records of our 637 fenestration operations and selected the poor candidates for this analysis. There have been a total of 44 poor candidates, or 7 per cent (see Chart I), of the total series of 637. After analyzing these results, if there is adequate air-bone gap I am a little more inclined to advocate surgery in these poor candidates.

CHART I.

INCIDENCE OF OPERATED POOR CANDIDATES.

0 — 100	4
100 — 200	5
200 — 300	8
300 — 400	8
400 — 500	5
500 — 600	11
600 — 637	3
Total	44

Chart I. In the total series of 637 fenestration operations there has been a total of 44 operations upon poor candidates, or an incidence of seven per cent per one hundred fenestration operations.

In a previous paper I defined the poor candidate as the otosclerotic patient with a gradually sloping and converging air and bone conduction audiograph, with the bone conduction for the frequency of 2,000 below the 30 db level. For the purpose of this paper I have extended this definition to include a few

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selected cases in which I was in doubt about the benefit of fenestration surgery.

These cases of otosclerosis have been classified into two large headings: Associated Nerve Deafness, and Independent Nerve Deafness. The associated nerve deafness is that usually seen in otosclerosis and is characterized by the gradual loss of bone and air conduction for all the higher tone frequencies, producing a gradually sloping air and bone conduction audiogram. The term independent nerve deafness is used whenever there has been some other type of nerve deafness combined with otosclerosis, as a high tone nerve deafness, or endolymphatic hydrops. Under the cases of the associated nerve deafness there have been four cases of the so-called malignant otosclerosis, with rapid loss of hearing the greater for the high tones, and pink promontory of the cochlea. The classification of these cases is shown in Chart II.

CHART II.

NERVE DEAFNESS IN OTOSCLEROSIS.

I.	Associated Nerve Deafness	33
	1-A. Malignant Otosclerosis	4
II.	Independent Nerve Deafness	
	1. High Tone	4
	2. Hydrops	2
III.	Osteogenesis Imperfecta	1
	Total	44

At the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society in 1954, I presented the results of the fenestration operation in the ideal candidate. It is easy enough to define the indications, and to advise these ideal candidates in which there is a greater than 90 per cent possibility of obtaining and maintaining a worthwhile hearing improvement through fenestration surgery. It is not so easy to evaluate and advise the poor candidates. These poor candidates must be individually selected, and only the surgeon himself can decide whether or not the patient will be happy with the partial result.

As previously stated, these candidates are usually advised as follows: "After a fenestration operation you will still need

a hearing aid for group gatherings. I would encourage you to continue the use of the hearing aid and to forget about your fenestration operation; however, I cannot deny you the partial benefit which you might receive through fenestration surgery". These patients are further advised they would have a 60 to 70 per cent possibility of obtaining a worthwhile partial hearing improvement. They are advised to tell their friends that they will still need to use an aid after surgery. Regardless of the result, they are advised to continue to wear the hearing aid for the first six weeks after they return to work, then to discard the aid only under those circumstances in which they know that they can get along.

ASSOCIATED NERVE DEAFNESS.

The 33 cases of associated nerve deafness have been divided into two groups. The first group of 25 cases in which the bone conduction at 2,000 was above the 40 db level, and the second group of eight cases in which the bone conduction at 2,000 was below the 40 db level. A few of these cases were unable to hear the bone conduction at the frequency of 2,000. In compiling these results to give the average, we have included the two cases who received less than 10 db improvement.

AVERAGE IMPROVEMENT.

Fig. 1 shows the average improvement in the cases with 30 db air-bone gap, and Fig. 2 shows the average improvement in these cases with less than 30 db air-bone gap at the frequency of 2,000. This composite audiogram (see Fig. 1) shows the average result in the 25 cases with bone conduction between the 30 and 40 db level at 2,000. These cases showed an average improvement of 33 db for the low tones, 32 db for the speech frequencies, and an average of 8 db for the higher tone frequencies. In greater detail this improved the hearing from the 57 db hearing level to the 24 db level for the lower tone frequencies of 125 and 250. The hearing was improved from the 70 db level to the 38 db level for the speech frequencies of 500, 1,000, and 2,000. The hearing was improved from the 79 db level to the 71 db level for the higher tone frequencies of 4,000 and 8,000. At the most critical frequency of 2,000 these patients experienced a hearing improvement from

the 73 db level to the 43 db level, or an average improvement of 30 db. This hearing improvement enables most of these patients to understand individual conversation. By and large they are grateful and happy patients. I have no hesitancy in

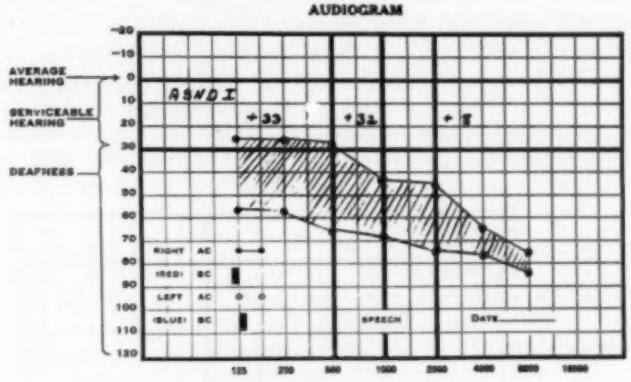

Fig. 1.

This composite audiogram shows the average result in the 25 cases of associated nerve deafness, with the pre-operative bone conduction between the 30-40 decibels level for the critical frequency of 2,000.

recommending the fenestration operation in these cases of moderately severe otosclerosis in which there is a 30 db air-bone gap for the speech frequencies.

The second composite audiogram (see Fig. 2) shows the average improvement in those patients with associated nerve deafness in which the bone conduction was below the 40 db level for the critical frequency of 2,000, and in which the air-bone gap was less than 30 db for this critical frequency of 2,000. These patients showed an average gain of 26 db for the low tones, 29 db for the speech frequencies, and 6 db for the high tones. In more detail, the hearing was improved from the 50 db level to the 24 db level for the low tone frequencies of 125 and 250. The hearing was improved from the 66 db level to the 37 db level for the speech frequencies of 500, 1,000 and 2,000; the hearing was improved from the 92 db level to

the 86 db level for the higher tone frequencies. Note on the figure, that the critical frequency of 2,000 shows the hearing was improved from the 78 db level to the 54 db level. With this sloping post-operative audiogram, these patients can hear about two-thirds of normal conversation. They must lip-read in order to understand the additional one-third of conversation. We advise all these patients to continue wearing the hearing aid, although with the concomitant loss at the frequency of 2,000 in the unoperated ear, most of them state that they can hear about as well in the operated ear as they can with the hearing aid in the unoperated ear.

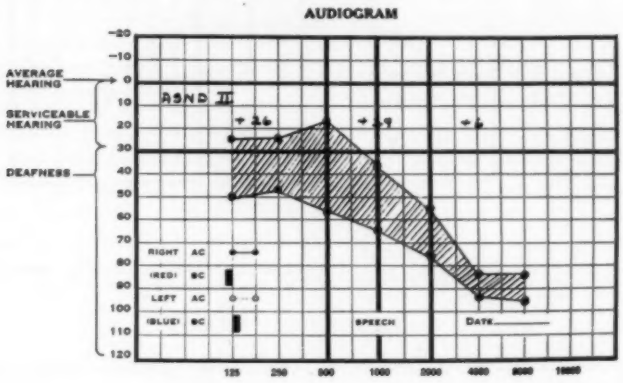

Fig. 2.
This composite audiogram shows the average improvement in those patients with associated nerve deafness where the pre-operative bone conduction was below the 40 decibels level for the critical frequency of 2,000. In Fig. 1 and Fig. 2, the composite average is based upon the best post-operative audiogram in the individual cases.

Included in the second group of associated nerve deafness are four cases of so-called malignant otosclerosis which deserve special consideration. By malignant otosclerosis we mean those cases of rapidly progressive hearing loss, with associated high-tone nerve deafness, and the pinkish promontory of the cochlea. One of these cases had about a 70 db hearing loss in both ears and was operated on in 1948. Three years post-operatively, she had maintained a 15 db hearing improvement in the operated ear, and had almost a complete loss of hearing in the unoperated ear. Today she can still wear the

hearing aid in the operated ear and has almost complete loss of hearing in the unoperated ear. It is my firm opinion that the fenestration operation has preserved the hearing for many additional years in this 23-year-old female.

Another of these cases is one of the most severe cases I have ever operated upon. This was done at the patient's insistence, and with thorough understanding of the limited possibilities. This patient had a 95 db hearing loss for the low tones and no bone conduction at the frequency of 2,000. This patient experienced a 27 db hearing improvement for the lower tones of 125, 250, and 500, and a 15 db hearing improvement for 1,000 and 2,000. Although this gives this very hard-of-hearing patient little subjective hearing improvement, it may be that in the years to come the fenestration operation will have preserved some of his hearing. This is equivocal surgery, and I do not recommend the fenestration operation in such cases; however, if I had such a deafness I would certainly have a fenestration operation. Not included in these averages is a fifth patient with severe "malignant otosclerosis", who stated that he would commit suicide if we did not operate upon him. Finally, at his doctor's insistence, we proceeded with a fenestration, obtained the anticipated poor result, and the patient subsequently hung himself.

INDEPENDENT NERVE DEAFNESS.

The cases of independent nerve deafness are divided according to type. First, there have been three cases with an almost complete loss of high tones in the presence of an otherwise flat air conduction audiogram, and good bone conduction for the existing frequencies. These patients showed a 26 db hearing improvement for the low tones; 22 db hearing improvement for the speech frequencies, and a 5 db improvement for the higher tone frequencies. Second, we have operated on two cases with probable hydrops of the labyrinth. At the time of the operation the operated ear was the poorest ear. In both cases the operated ear is now the better ear. In one case the patient has good speech perception in the operated ear, there has been some decrease in the tinnitus in the operated ear. In the other case there was a 20 db hearing improvement, bringing the hearing in the operated ear to about the same level as

that in the unoperated ear, but no worthwhile improvement so far as the patient was concerned. This is a disgruntled patient whom I have not seen in recent years. I have no conclusions to offer in those isolated cases where there is an associated endolymphatic hydrops in the presence of otosclerosis.

CLOSURES.

There have been no known closures in this group of 44 cases. In the last 300 cases there have been less than 2 per cent known closures. The fenestra have been made with sharp hollow ground margins under irrigation and under 7, 15, and now 40-power magnification.

COMMENTS.

The review of these poor candidates for fenestration surgery has revealed that the fenestration operation is of greater value than I had anticipated. In otosclerosis with the typical associated nerve deafness it is my opinion that the fenestration operation is a thoroughly worthwhile procedure, even though we can anticipate only a partial restoration of hearing. This opinion is substantiated by an average improvement of approximately 30 db for the speech frequencies in these cases of associated nerve deafness.

The patients with moderately severe or severe otosclerosis are very grateful for their partial hearing improvement. If the operation enables them to understand normal conversation at three to five feet, they are very pleased with their restoration of hearing.

The non-fenestrating otologists and the general public are inclined to be critical of the fenestration operation in these cases, because the patients have had the operation and they are still hard-of-hearing. The plea of this presentation is for tolerance. When one considers that these patients were profoundly deaf and completely out of auditory contact with the world, and that they have been improved to the ability to understand most individual conversations at the distance of a few feet, and that they can hear most ordinary noises, this seems to me to be very worthwhile surgery.

SUMMARY.

1. Forty-four fenestration operations on poor candidates have been selected from a total series of 637 fenestration operations and the results analyzed.

2. The poor candidates were subdivided into otosclerosis with typical associated nerve loss, and into otosclerosis with independent nerve deafness.

3. The cases of associated nerve deafness have obtained an average gain of about 30 db in the speech frequencies, improving their hearing from auditory isolation, to contact with the world.

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509 Bayshore.

THE NEWCOMB AWARD.

At the Seventy-seventh Annual Meeting of the American Laryngological Association held at the Seignory Club, P. Q., Canada, May 13-14, 1956, the Newcomb Award of the American Laryngological Association was presented to Dr. Dean M. Lierle, of Iowa City, Iowa, "for his significant contributions to Laryngology and Rhinology and to the American Board of Otolaryngology."

THE ROLE OF THE CRICOTHYROID MUSCLE IN TENSION OF THE VOCAL CORDS.

An Experimental Study in Dogs Designed to Release Tension of the Vocal Cords in Bilateral Recurrent Laryngeal Nerve Paralysis.*

LOUIS M. FREEDMAN, M.D.,

Boston, Mass.

That a way would be found to free the patient with bilateral recurrent laryngeal nerve paralysis from the lifetime use of a tracheotomy tube has been in my mind and hope for many years. This has been the ambition of others also, and many operations, such as the King, Kelly, and Woodman[1] operations, had the same purpose; however, they involved removal of the arytenoid cartilage on one side, changing the position of the vocal cord on that side, working in a difficult, deep corner in a more or less long operative procedure—or of moving one cord away from the median line. They presumed the acceptance in more than mere principle of the Semon[2,3] law, which states that "in bilateral recurrent laryngeal nerve paralysis adduction prevails longer than abduction because the posterior cricoarytenoid muscle, which controls abduction, fails before the cricoarytenoid lateralis, which controls adduction."

Murtagh and Campbell[4,5] lent support to this law by observation in animal experiments that the fibers of the adductor muscles were greater in size and number and more heavily medulated than those of the abductor muscles. General acceptance of this law led to the development of the King, Kelly, and Woodman[1] operations. Clerf and Baltzell[6] questioned the validity of the Law on the ground that the interarytenoid muscle is innervated by the external subdivision of the superior laryngeal nerve as well as by the recurrent laryngeal nerve; however, Lemere[7] showed that this branch of the superior laryn-

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geal traverses the muscle only to supply the underlying mucous membrane. Semon's observations were made largely in cases of injury to the nerves by tumors, aneurisms, and other conditions which produce gradual and varying degrees of imbalance in power between the abductor and adductor fibers; but the Law does not explain the paramedian position of the cords under all possible conditions and especially after complete surgical division of the nerves. It seemed obvious that it made little difference which muscle was stronger in its activity if both muscles failed in their activity at the same time.

During the years 1932-1934 I[12] had occasion to observe a fairly large number of recurrent laryngeal nerve paralyses following total thyroidectomy. These cases were done for the purpose of reducing the metabolic rate and lessening the work of the heart in chronic heart disease, in a study by Blumgart, Levine, and Berlin.[12] Most of the cases I observed that had a recurrent laryngeal nerve paralysis were unilateral and required no tracheotomy; however, the paramedian position of the cord in all cases was striking. The more I pondered on this the less convincing the Semon explanation seemed. I could well understand that, in a gradual pressure by tumor or aneurism on a recurrent nerve, paralysis of the muscles might occur in an uneven manner; but when both muscles were inactivated at the same time the resultant position of the cord should not favor either abduction or adduction.

In 1883 F. H. Hooper,[8] of the Massachusetts General Hospital, reported in "Experimental Researches on the Tension of the Vocal Bands" that the cricothyroid muscle was innervated by the external branch of the superior laryngeal nerve, fixed at the lower border of the thyroid cartilage and attached to the upper border of the cricoid cartilage. In contraction he observed that this muscle pulled up the cricoid cartilage to tense the vocal cord. He also explained the importance of the blast of air from below and the use of the extrinsic laryngeal muscles in causing higher degrees of tension, enabling the singer to reach higher notes. This was the first instance in which serious attention was paid to any factor other than the recurrent laryngeal nerves in relation to tension and the mobility of the vocal cords.

It seemed best to study this problem from a fresh point of view, leaving Semon's Law in abeyance for the time being; therefore I undertook a study of many postmortem larynges. It was quite evident that the vocal cords now were widely open and gave apparently sufficient space for respiration. This was really the cadaveric position. In contradistinction, in the living, when only the recurrent laryngeal nerves are bilaterally severed, the cords are in the paramedian closed position. This is definitely not the cadaveric position and implies that another nerve or muscle must be concerned in keeping these cords in tension and in the paramedian position. A further study brought realization of the fact that this tension was assisted by the pivotal action of the inferior cornus of the thyroid cartilage, attached by a synovial joint to the cricoid cartilage on each side, assisted in tensing the vocal cords by contracting the cricothyroid muscle in bringing up the cricoid cartilage toward the lower border of the thyroid cartilage, and this muscle, the cricothyroid, was innervated by the external branch of the superior laryngeal nerve.

Woodman[1] also seems to have realized this, because in his procedure he also released this attachment. A postmortem larynx, in contradistinction to a bilateral recurrent laryngeal nerve paralysis in the living, shows the vocal cords to be wide open instead of closed. It seemed to me that it would be wise to reconsider the innervation of all the intrinsic muscles and nerves of the larynx to determine all the factors concerned with the opening, closing, and tensing of the vocal cords.

Muscle.	Nerve.	Action on Vocal Cord
Cricoarytenoid Posterior.	Recurrent Laryngeal.	Abduction.
Cricoarytenoid Lateralis.	Recurrent Laryngeal.	Adduction.
Cricothyroid.	External Branch of the Superior Laryngeal.	Tension.
Arytenoideus.	External Branch of the Superior Laryngeal and partly by the Recurrent Laryngeal.	Tension.
Thyroarytenoid.	Recurrent Laryngeal. Rest of the Superior Laryngeal.	Abduction. Sensory.

A study of these nerves, muscles, and their action on the larynx, showed, as does this chart, that tension was not merely a function of adduction, but also that the cricothyroid muscle must be an important factor in the paramedian position. Murtagh and Campbell[4,5] on the one hand and Terracol[9] on the other, demonstrated to their own satisfaction that the cricothyroid muscle, when tensed, was a very strong adductor at the same time. When the recurrent laryngeal nerves are severed without any other reason for tension the balance between abduction and adduction cannot be altered in favor of either; but relaxation of the vocal cord without tension ought not to allow the paramedian position, but rather relaxation only.

A study in a series of dogs was undertaken to test the hypothesis that if tension was altered or lost by severance of the external division of the superior laryngeal nerve, or better, lost by prevention of tension in any form by severing the cricothyroid muscle and detaching the inferior cornua of the thyroid cartilage from the cricoid cartilage so that no pivotal action on the cords could be attained, it might be possible to keep the cords sufficiently open so that the tracheotomy tube might be rendered unnecessary.

Under general anesthesia, both recurrent laryngeal nerves were severed and, when the cords were found to be in the median line, a tracheotomy was performed and a tracheotomy tube inserted. After two to three weeks the tissues were healed and the vocal cords were found to be in the paramedian position. Then, in another procedure, under general anesthesia, the cricothyroid muscle was severed from the lower border of the thyroid cartilage and the upper border of the cricoid cartilage and the inferior cornua severed from the thyroid cartilage on each side. After two more weeks it was found possible to remove the tracheotomy tube, because the vocal cords were sufficiently open to allow proper breathing without it. Various incisions were tried, especially to be sure of the location of the recurrent laryngeal nerves. Eventually it was found that a horizontal incision allowed all the necessary procedures to be carried out with the least amount of trauma and disturbance of the tissues, since after the horizontal incision through the skin the musculature under it could be

easily manipulated in any direction. We tried the effect of merely severing the cricothyroid muscle and the inferior cornu on the same side. Although this succeeded it seemed that the complete work on both sides would carry greater assurance of success and nothing would be lost.

During this study my attention was called to an article by N. D. Fischer[10] entitled "Preliminary Report on the Application of the Motor Function of the Superior Larnygeal Nerve." In this paper Dr. Fischer suggested that the motor division of the superior laryngeal nerve in man has the same function as in the dog and that in dog experiments in which he had cut the right recurrent nerve and the right superior laryngeal nerve, the right vocal cord remained in the so-called cadaveric position and allowed for a good glottic chink. Since this procedure destroys laryngeal sensation, it would seem to be more desirable to cut the external branch of the superior laryngeal nerve alone, or to sever the cricothyroid muscle so as not to interfere with sensation. Moreover, as shown by Dilworth[11] and others, the free anastomoses between the branches of the superior and inferior laryngeal nerves would possibly make it difficult to find the external branch of the superior laryngeal nerve or to prevent complete loss of tension, which the anastomoses might defeat. For that reason we decided that it would be wiser to remove the cricothyroid muscle so as to attain as near complete loss of tension as possible. In all, fifteen dogs were used.

May I here describe a few of the procedures carried out on these dogs, and their purpose.

Experiment No. 1. Under general anesthesia, complete severance and removal of the cricothyroid muscle from the superior margin of the cricoid cartilage and from the lower border of the thyroid cartilage, and at the same time, severance of each inferior cornu from the thyroid cartilage and detachment from their attachments to the lateral surfaces of the cricoid cartilage. Ten days later examination of the larynx showed complete abduction but incomplete adduction, so that the vocal cords could not approximate evenly in the median line. This lent support to the presumption that tension was

destroyed to the degree that the cords did not approximate at the median line.

Experiment No. 2. Only the left recurrent nerve was severed and the cricothyroid muscle removed on the right. Examination of the cords showed left recurrent paralysis, with the right overacting beyond the median line. One week later the right recurrent laryngeal was severed and the cricothyroid similarly treated as in the first operation. There seemed to be so much space at the end of the procedure that tracheotomy was not done—with the result that edema caused obstruction and no opportunity for continued respiratory activity—so that two days later the dog was found dead.

Experiment No. 3. The entire cricothyroid muscle was removed together with both inferior cornua of the thyroid cartilage and severance of the right recurrent nerve. Postoperatively, the right cord was immobile but was moved away from the median line, and the left cord was mobile but relaxed away from the median line. Tracheotomy was done. Ten days later the tracheotomy tube was extruded because it was blocked with secretion, but the tracheal opening was free and respiration was unimpeded. The tube was reinserted but removed after a few days because there was no edema of the cords and there was 5 mm. of space between them. This dog was observed for two months, and had no respiratory difficulty.

Experiment No. 4. The left recurrent laryngeal nerve was severed and the cricothyroid muscle removed, together with both inferior cornua of the thyroid cartilage. Postoperative examination showed left paralysis. No tracheotomy was done. One week later the right recurrent nerve was severed, but no tracheotomy was done, because there was no evident edema from the previous operation. This dog was observed for a number of months and no respiratory difficulty was noted.

Experiment No. 5. Both recurrents were severed and tracheotomy tube placed. Both cords were in the paramedian position. Ten days later the cricothyroid muscle was removed and the inferior cornua of the thyroid cartilage, also. The tracheotomy tube was left in place. One week later there was 6 mm. of space between the cords. The tracheotomy tube was removed, and there was no respiratory difficulty.

Experiment No. 6. Both recurrents were severed and only the left half of the cricothyroid severed, and dog was tracheotomized. Both cords were immobile, but there was 4 mm. of space between them. The tracheotomy tube was removed one week later, and respiration was unimpeded.

Summary: On the principle that abduction, adduction, and tension are the three main factors concerned with the motility of the vocal cords and free respiration, a series of experiments was carried out on dogs, first, to sever both recurrent laryngeal nerves and thus insure bilateral recurrent laryngeal paralysis. Tracheotomy was then carried out. Later, the cricothyroid muscles were detached from the lower border of the thyroid cartilage and the inferior cornua of the thyroid cartilage severed from their attachments to the lateral surfaces of the cricoid cartilage. When, by observation, it was evident that the glottic chink was wide enough for normal respiration, the tracheotomy tube was removed and the wound closed. The dogs were able to breathe freely without apparent discomfort thereafter. It is hoped that this method may be of use in human beings, and make unnecessary more complex procedures involving removal or displacement of the arytenoid and sewing back of the vocal cords. The procedure herein described is so simple that it would seem to merit trial in human beings.

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HEMICRANIA—OR ONE-SIDED SPHENOPALATINE ETHMOID HEADACHE.*

WM. H. TURNLEY, M.D.,

Ocala, Fla.

The Greek word "hemicrania" means half-head, and the term was first adopted by Galen (201-137 B. C.) but, in the course of time, underwent considerable etymological ill-treatment, until it was corrupted from "hemicrania" to "emigrana", then "migrana", and finally to the French term "migraine". I am not going to discuss the other side, for this headache is predominantly on one side; although, after a long attack, it may become a generalized headache on both sides, or the patient just does not care, and says it is all over the head. The origin seems to be definitely located in the sphenopalatine ganglion region of the nose, between the posterior ethmoid cells and nasal septum. The syndrome may radiate from this spot to the entire side of the body in extreme cases, simulating the whole gamut of headaches from tic to epilepsy.

One reason I chose this subject at this time is because the literature has gone overboard with the emphasis on "vascular headache", but I still think a focus of infection plays an important role, whether it be bacteria, virus or their mutations; and I might say in the beginning, I have no use for foci of infection anywhere, at any time, and that goes for germs in the human body, weeds in the garden, and crooked politicians in government.

Another reason I chose this subject may be a local one, for I practiced otorhinolaryngology in New York City and Connecticut for 25 years, then suddenly moved to Ocala, Fla. In the past four years, I have seen more of this type of headache here in this part of the South than I did in a metropolitan northern area in ten years. I believe it is due to the predominance of the Anglo-Saxon or Caucasian people in this region,

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who have a high, narrow nose with pressed-in wings, in contrast to the mixture of races of people who have a broader nose.

In reviewing the literature on this subject over the past decade, I find there are very few articles concerning the sphenopalatine syndrome, but so many volumes have been written on histaminic cephalalgia, endocrine headache, trigeminal neuralgia, tension headaches, myalgia, allergic headaches, vascular phenomena, and atypical everything, that it is, at least, confusing. I have written this paper from my own practice and experience, and if some of you disagree with it, that's good. We all disagree on some things, and the Bible says there will never be a time when any two men are of the same accord.

A review of the anatomy of the sphenopalatine area is adequately described in Morris' *Anatomy*. The nasal fossae are two irregular cavities situated on each side of the median vertical septum. Each fossa communicates with the sinuses of the frontal, sphenoid, maxilla, and ethmoid bones.

The roof is horizontal in the middle but slopes downward in front and behind. The anterior slope is formed by the posterior surface of the nasal bone and the nasal process of the frontal; the horizontal portion corresponds to the cribriform plate of the ethmoid and the sphenoidal concha; the posterior slope is formed by the inferior surface of the body of the sphenoid, the ala of the vomer, and a small portion of the sphenoidal process of the palate. The sphenoidal sinus opens at the upper and back part of the roof into the spheno-ethmoidal recess, above the superior meatus.

The septum is the median wall which is usually deflected from the middle line to one side or the other and occasionally has large spurs. It also may contain ethmoid cells superiorly. The lateral wall is the most extensive and the most complicated on account of the formation of the meatuses of the nose. The superior meatus, the shortest of the three, is situated between the superior and middle nasal conchae, and into it open the orifice of the posterior ethmoidal cells and the sphenopalatine foramen.

The nerve center is situated in the sphenopalatine, or Meck-

el's, ganglion, and is associated with the maxillary nerve. The ganglion is a reddish-grey body and measures about 5 mm. It lies deeply in the pterygopalatine (spheno-maxillary) fossa at the lateral side of the sphenopalatine foramen and in front of the anterior end of the pterygoid (Vidian) canal. It is attached to the maxillary nerve, from which it receives its sensory root, and it is connected with the Vidian nerves, which furnish it with motor and sympathetic filaments.

The Vidian nerve is formed by the union of the great superficial and deep petrosal nerves in the foramen lacerum to the sphenopalatine ganglion. The Vidian nerve is joined by a sphenoidal filament from the otic ganglion, and it gives branches to the upper and back part of the roof and septum of the nose and to the lower end of the Eustachian tube.

The anatomical and clinical evaluation of head pain attributed to the sphenopalatine ganglion was very carefully studied by Greenfield Sluder in 1908. He thought that the origin of this type of headache is by extension from the posterior ethmoid and sphenoid sinuses and said, "In no other part of the body is a sympathetic ganglion or sensory ganglion so exposed to surface influences." Of course, headache may have more than one cause, but, in this particular type there is swelling and infection whether it be of bacterial or virus origin, or both. The pathologic basis may be in dispute, but most students believe that a disturbance in the cerebral vasomotor mechanism underlies all cases. This may be just another symptom of the original sphenopalatine disturbance, for wherever there is swelling, from infection or not, there is pressure on the nerve end organs, which is transmitted as pain.

Sluder also thought that the sphenopalatine ganglion is not separated from the nasal cavity by bone; therefore, many anatomists are not willing to accept this as the absolute interpretation, and that leaves the anatomic solution of this referred pain problem still unsettled. Rather than get into this controversy, I refer you to articles by Robert B. Lewy, in the *Journal of Laryngology and Otology*, Vol. 65, No. 1, Jan., 1951; Dr. David R. Higbee, *Transactions of the American Academy of Ophthalmology and Otolaryngology*, 52: 283, March, 1948.

I am merely trying to tell you the clinical results I have obtained from a careful and thorough submucous resection, especially of the posterior and superior nasal septum in this particular type of headache.

The first sign of this neuralgic syndrome may start as a stuffy feeling in the nose, with all the symptoms of a cold. The attack may come on rather suddenly, following a change of atmospheric pressure, temperature and humidity. This dull feeling in the nose is always on one side at first, the eye aches deep in the back, and the patient may think he needs glasses or a change of lenses. The pain may be across the bridge of the nose or a tenderness of the upper inner angle of the orbit of the eye, or sharp pain in the temple. There is often an area on top of the head which is sensitive even to touch. Many cases have a definite tender spot just back of the mastoid process, as Sluder describes it, 5 cm. post to mastoid tip. The ache is always in the back of the head and occiput, neck and shoulder and down the deltoid muscle of the arm to the elbow, or even to the fingertips.

Some describe a low-grade ache over the maxilla, and along the Eustachian tube; but the most common course is around the eye, cheek, temple, top of head, occiput, back of neck and shoulder. Sometimes, this syndrome will last for a few days and clear up voluntarily under certain conditions. It may last for several years at a time, as a constant, low-grade ache; again, there may be excruciating attacks, and the patient may become almost hysterical, with nausea and vomiting. These patients are sure they have a brain tumor, and may contemplate suicide. I believe trigeminal neuralgia, especially of the ophthalmic and maxillary branch, is just a more drastic form or different manifestation, of this same syndrome; likewise, herpes zoster ophthalmicus and oticus are of the same origin.

After taking a further history of these cases, there is generally a story of always having a cold, and eventually the patients may tell you that they have lost their sense of smell (anosmia). Then you know they have a definite ethmoid sinus infection of many years' duration. A post-nasal drip is nearly always a forerunner, and it may not necessarily be pus; later, there may be a bloody discharge.

On examining the nose, the casual observer may say the patient has no crooked septum or sinus condition; but, after shrinking the turbinates, a closer examination, far back and high up in the post-ethmoid region, will show you the septum and turbinates are stuck together so tightly that a probe can not be passed between them. The septum may be swollen several times thicker than normal, but as soon as you separate it from the superior turbinate with a probe, tipped with cotton soaked in 10 per cent cocain, the patient says, "Oh! I can breathe now; my whole head feels clearer, and that ache in the back of my neck is gone."

Here again in this region, that old adage "Symptoms are more important than signs" holds true, for an X-ray film seldom shows anything unless there is a definite bacterial infection in the posterior ethmoid cells. The sphenoid sinus does not seem to be involved as often as was once thought. I believe transillumination gives just as much information as an X-ray does in these cases, and it can be done more quickly. Seldom is there any temperature, except in an acute fulminating condition.

There is quite often a Eustachian tubal catarrh, with a slight loss of hearing for the low 128 fork, bone conduction greater than air conduction. Weber test lateralizes to the affected side.

Children do not seem to have this type of headache so much as adults. This is probably because they are more flexible, not so sensitive to pain as adults, don't know how to describe their condition, or that the growing process has not developed the septum to the final stage causing pressure; but when the headache is present, it should be corrected by a submucous resection at whatever age they are. I have often wondered whether a child delivered by forceps is more likely to have a depressed bridge of the nose, thereby deflecting the septum, than those delivered without instrumentation. Patients with a high acute-angle, bony palatine arch, nearly always have a deflected septum.

Trauma late in life, from a blow that may break the bone or distort the septum, does not seem to be an etiological factor for this headache. I believe the cause is more of a develop-

mental origin. A thermostatic imbalance, along with a change of barometric pressure and humidity, may be an exciting physical factor, while a disturbed endocrine balance may be an exciting physiological factor. In other words, anything which causes the turbinates to impinge against the septum permanently and block off the ethmoid air drainage, may start the syndrome. Males and females are about evenly divided as to sex origin.

There seems to be two schools of otolaryngologists: those with a medical outlook, and those with a surgical outlook. We need both, for there is still plenty of surgery to do, and a whole new world of therapy to come. Some day we will find out what a cold or sinusitis really is, but, for the present, I think the first running nose a baby has, at the age of a few months, is his first cold or ethmoid sinusitis, and from then on it may progress, or subside, depending upon his natural resistance and the various factors that influence him. The cold may be a virus that just gets the soil ready for the seed, so to speak. Then the secondary invaders of bacteria gain entrance, and one has a real bacterial sinusitis; but all this is a vicious cycle, and as the baby grows, so does the septum of his nose, and if this septum does not have room to expand, it will bend to one side or the other, and block off the ostia of the ethmoid sinus. The nose must have free circulation of air all the time, just like the rooms in a house, for if there isn't good air circulation, things get stale and mouldy. The ostea of the posterior ethmoid cells are small, and it does not take much swelling of the mucosa, or pressure from a curved septal wall, to block them.

Like the dirt in the eye of a needle, it does not take much dirt to block the eye, but until you get this little amount out, the needle is useless for sewing, regardless of the other dirt around the needle. When you look in a nose and see an obvious large deviated septum or spur on the floor, this is not nearly so bad as that gentle little compensatory curve of the septum, situated far back, and at the top of the septum. You can hardly see this curve, especially if the turbinates are swollen; but, if you shrink the turbinates with a solution of cocain, then you can see this deflection, or obstruction. As I said before, a lit-

tle blockage in this area does more harm than the big, obvious deviation below. The patient seems to get plenty of air under the inferior and mid-turbinate, but yet he says he has a feeling of stuffiness up high, and he may complain of a musty smell. This is one of the most sensitive areas in the body, and if you don't believe it, just try to probe it on yourself. It is amazing how excruciatingly painful just the slightest pressure can be. That is why so many doctors do not get this area separated, for the patient won't let him place the probe there; but it is imperative that you cocainize this area, so wait for a minute or two until the cocain takes effect, and then place a fresh probe, with a little 10 per cent cocain, further up. When you hear a clicking sound, like that of two glued pieces of paper pulled apart, then you know you have let air into this area, and the patient gives a sigh of relief and says, "Why this is the best I have breathed through my nose in months." Almost immediately, the pain on the top of his head, or behind his ear, or back of his neck, is relieved. It is very spectacular, and this is a good sign you have hit the spot.

Sometimes just one treatment is sufficient to relieve a patient for several months, or you may have to do this every day or so for several times. He can be given a mild spray, of chloretone inhalant, or your own pet Rx, but the best way to give this patient permanent relief, is to do a thorough submucous resection of the septum.

It is not necessary to describe this operation, except to say you must get that little curve up high and back in the ethmoid region, and to do that it is necessary to resect the septum below and front. Many beginners do a partial submucous resection, get a perforation, and stop, or think they have done a complete submucous resection. Not so; you might just as well have done nothing so far as relieving this type of headache. It is not a difficult operation to do, but it is a tricky one; like a game of golf, you must be accurate and complete the stroke all the way through to shoot par.

When a patient tells you he has had a submucous resection operation, don't believe him; look for yourself, even if it has been done by the best surgeon in "Podunk", or even if it has been done twice. There is one thing that seems to be avoided in the literature on surgery, and that is the criticism of poor

or incomplete surgery. We all know that there are more poor tonsillectomies done than any operation in our specialty, but there are some poor submucous resections done, too. I have done this operation after it had been attempted twice before, and yet I got good clinical results. It is not fair to the patient, it is not fair to the operation, and it is not fair to the practice of otolaryngology to let a poor result remain. Yes, it is difficult to convince these patients that the end-result has not been accomplished the first two times, and that you can do it this time, but it can be done.

A submucous resection should always be done before performing an ethmoid or sphenoid operation. It can do no harm anyway, and it gives room for the turbinates to function more freely. Do not disturb the turbinates unless absolutely necessary, as they have a definite physiological function to perform, as radiators and humidifiers. Even if the turbinates are slightly polypoid, they will return to normal in a year's time, if the nose is given good air drainage by the submucous resection.

This operation is not intended to cure the ethmoid sinus infection *per se,* but it does allow better ventilation of this whole area, which will in turn allow nature to drain and heal whatever infection there is. It allows the turbinates to expand and retract normally, thereby performing their important functions; if the turbinates do swell, they can now press against a flexible curtain, rather than a rigid wall, thereby relieving the headache. The operation has nothing to do with impairing the structure of the nose if done properly. It allows freer access to treat the nose, in case of subsequent trouble, and it does no other harm that I have been able to detect; furthermore, if there is a large deviated septum, a good submucous will, to some extent, allow a crooked external tip to come back into line. Do not forget to take out any adenoids that may be present, too, as this may be just the added stroke to give better results.

Some patients may not get the desired results for six months. It is like removing the obstruction in the stream that drains the springhead, which, when backed up, causes a swamp. This swamp may not drain out completely the next day, as there are

a lot of factors that come into play. When the swamp dries up, the turtles, mosquitoes, and water lilies die, so when the ethmoid sinus gets aerated and drained, the streptococci and other germs diminish too. I believe it was Hippocrates, years ago, who advocated good drainage and aeration, wherever there is blockage to infection.

Sometimes the patient may have a definite chronically infected ethmoid condition. If so, one has to use his judgment as to whether to take off the superior turbinate, or do a partial ethmoidectomy. I have a great deal more respect for the ethmoid cells than I did as an intern. Now I leave them alone, but if they are definitely destroyed and cystic, it may be necessary to remove or open them; for an infected hidden ethmoid cell may suffice to keep in action the entire toxic syndrome, if it cannot have drainage.

A submucous resection is easier to do under local anesthesia, rather than under general anesthesia, but children and some adults require a general anesthesia. I believe this operation should be done early in life rather than late, as formerly advocated. No harm can be done after 10 years of age; also, I think all asthmatic patients should have this operation done, whether they have headache or not.

As for the medical treatment of this type of headache, I am sure you are familiar with the best of modern drugs. Naturally, we want to give these people relief, but no drug seems to work too well, not even demerol or morphine, but cocainization of the sphenopalatine ganglion area will relieve most patients of pain for an hour, if not for a longer period of time. All of my extreme cases have had the "pharmacopoeia thrown at them"—histamine de-sensitization, anti-histamine, estrogens, nicotinic acid, vitamins, diets, ergotamine tartrate, amphetamine, cortone, and the vasodilators. Probably as good as any is a cafergot suppository, a dark quiet room and a warm bed. A drink of bourbon is better than a cigarette any time. The best prophylactic I know is to avoid getting chilled, or a cold, or an emotional upset.

I must mention virus as a type of infection in this area to be treated, since recent research has already produced an astonishing array of hitherto unknown viruses. At the last offi-

cial count, 356 new virus strains have been found in the course of polio research; how many more exist is anybody's guess. They arrive so fast that researchers have found no time to keep up with them, and have no idea what most of them do. Dr. Joseph Melnick, of Yale, has given them a name that has caught on—"orphan viruses", and refers to them as "viruses looking for a disease."

Doctors Huebner, Rowe, Ward and Parrott, of the National Microbiological Institute, have come up with a virus they named RI (for respiratory infection) 67. Now the picture has continued to grow, until there are eleven related but distinct virus types that have been found in the nasal cavity. They cause many diseases of the upper respiratory region as "pink eye", sore throat, tonsillitis, and a stuffy nose with headache. Already a virus for herpes zoster has been isolated. Like all true viruses, however, there is no preventive yet, except the brief protection from shots of gamma globulin, although a specific vaccine is theoretically possible.

In conclusion, I think the treatment "par excellence" is relieving the pressure and allowing ventilation and drainage in the sphenopalatine ethmoid area, by a careful and thorough submucous resection of the nasal septal cartilage, especially posteriorly and superiorly. Even this is not a panacea, for there will still be some with a headache, but I hope you are not one.

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Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Paul H. Holinger, 700 No. Michigan, Chicago 11, Ill.
Vice-President: Dr. Jack Allan Weiss, 109 No. Wabash Ave., Chicago 3, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting: May 26 - June 2, 1956.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.

Vice-President: Dr. George E. Bradford, Winston-Salem, N. C.

Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.

Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Norman Eaddy, Sumter, S. Car.

Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.

Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.

Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.

President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.

Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A. R. Hollender, 605 Lincoln Road, Miami Beach, Fla.

Vice-President: Dr. Curtis D. Benton, Jr.

Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.

Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edwin G. Grafton.

Vice-President: Dr. L. A. Furchgott.

Secretary-Treasurer: Dr. Hal W. Maxwell.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Leland R. House, M.D.

Secretary-Treasurer: Sol Rome, M.D.

Chairman of Ophthalmology Section: Stephen J. Popovich, M.D.

Secretary of Ophthalmology Section: Richard Kratz, M.D.

Chairman of Otolaryngology Section: Harold Boyd, M.D.

Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.

Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington

Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.

Meeting: April 7-11, 1957.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Hon. Chairman: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles 57, Calif.
Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. S. Gorrell, 110 Medical Arts Bldg., Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: June 6-7, 1956, Chateau Frontenac, Quebec, Canada.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

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Vice-President: Dr. Jorge de Cárdenas.
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SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
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MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

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Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
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Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
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BUENOS AIRES CLUB OTOLARINGOLOGICO

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SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

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Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
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SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

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SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID

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PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida de Liberdade 65, 1° Lisbon.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA

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2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1.°, Lisboa.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

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Tesorero: Dr. Juan Manuel Pradales.
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ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filno Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Apartado Postal 21815
Mexico, D. F.

Honorary President: Dr. Ricardo Tapia y Fernández.
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Secretary: Dr. Luis Vaquero.
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Treasurer: Dr. Rubén Mingram Camargo.

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(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

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Librarian: Dr. P. G. Gerlings, J. J. Viottastraat 4—Amsterdam.
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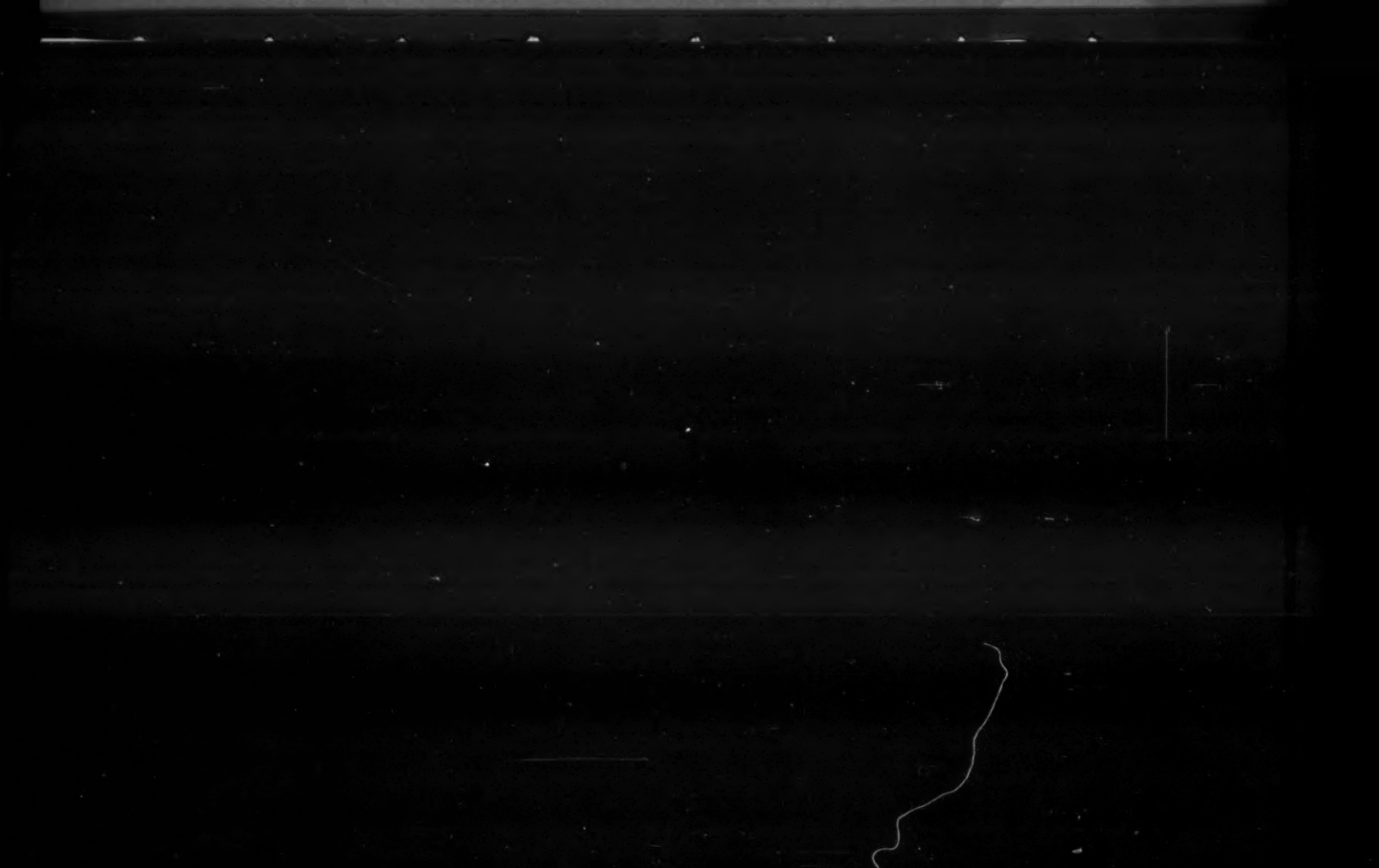

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